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PRESENT STATUS OF TREATMENT OF DETACHMENT OF THE RETINA

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The present importance of the treatment of detachment of the retina is of the utmost interest to all oculists.

The most pronounced skeptics who doubted the results mentioned five years ago, when cures were cited in an average of 25 per cent of the cases, will probably still doubt when they hear it said today that cures are obtained in more than 50 per cent of the cases; but they cannot fail to recognize that enormous strides have been made in the treatment of this grave condition and that further advances will be instituted in it.

The oculists who accepted the facts demonstrated five years ago and followed the technics recognized as best have been confronted with a series of new operative methods which, changing almost every year, have confused them as to the course to be followed, obliging them to vary their instrumentation and technic. At present, experience permits a more precise showing of operative indications and recognition of the more pertinent material, conferring thus great facility on ophthalmologists grounded in this treatment and making it accessible to those who have not yet used it.

Finally, those who first carried out these operations have seen during the immediate past a great many cases of detachment of the retina, affording an abundance of material for study, and since this is due to the confidence which their colleagues have placed in them, the fact cannot be mentioned without tendering to these colleagues a most sincere and profound gratitude. This material has not only permitted those interested in the matter to perfect the treatment of detachment of the retina, but also to study the disease per se, its etiology, pathology and evolution.

The basis of the treatment of detachment of the retina continues to be the same; that is to say, obstruction of the tears in the retina which almost always exist in this condition. When, without closure of the tear, one is successful in producing adhesion of the retina surrounding it to the choroid, cure of the detachment or rather reattachment of the retina to the choroid is also obtained. Therefore it is necessary to interrupt the communication between the vitreous and the subretinal space which takes place through the tear.

Toward that end are aimed the operations proposed in the last few years for the treatment of detachment of the retina: the use of the thermocautery suggested by Gonin, the use of the galvanocautery which many others have adopted, the modification by Vogt, the scleral trepanations of Guist, the delimiting method of Lindner, the employment of diathermy in its different modalities (Weve, Šáfár) and the combinations of these different methods.

Whatever method is employed (later I shall take up the indications for each of them), the operation must be preceded by a clinical study of the case, for side by side with the simple cases in which the technic to follow is easy to determine and to execute, there are those which require a more profound study based on a complete examination of the ocular fundus, which at times is difficult because of opacities of the transparent media or because of unfavorable conditions peculiar to the case.

OPERABLE CASES

Since detachment of the retina was formerly a disease little less than incurable, and since the percentage of cures recently obtained is very high (in a recent series of cases, it exceeded 70 per cent), one might assume that in practically all cases operation should be attempted; but this would not be true, for there are cases in which the condition even from the first days is inoperable. A very important factor is the age of the detachment. In general, the longer the time that has elapsed, the more difficult it is to obtain reattachment of the retina, although the primal factor is the anatomopathologic state of the detached area, there being cases with favorable aspects after a duration of several months to a year in which complete reattachment is obtained. In these cases, although vision improves and is stabilized, the retina has lost some of the attributes of visual acuity and, above all, that of adaptation for light and dark.

The factors favorable for a successful operative result are: a good condition, general and local; the etiology of trauma; an absence of local reaction, and, particularly, little lapse of time since the occurrence of the detachment—less because of the elapsed time than because of the anatomopathologic conditions, which become progressively aggravated. In the beginning, the tears are small and generally the retina is little detached, although there are cases of superior detachment in which even from the first day the bulging of the retina is very pronounced. In like manner, in the beginning the vitreous is usually clear, but as time passes it becomes cloudy as a result of the inflammatory process which develops in the choroid on its coming in contact with the vitreous, which has the effect of a foreign body. A very frequent exception to this rule of the transparency of the vitreous in the early days of detachment must be noted: the apparent production of detachment through

hemorrhage into the vitreous, due probably to rupture of vessels at the level of the tear. Thus it is seen frequently that the primary diagnosis is that of hemorrhage into the vitreous because of the view of blood in it (which certainly prevents one's seeing anything further) or that of exudative choroiditis because of the presence of a turbid grayish zone.

Developments of the latter type constitute the unfavorable conditions for operative treatment, and only in case the hemorrhage or turbidity of the vitreous becomes very much localized should intervention be attempted, and then it will always be done blindly and with less probability of success than if one waits for several days until the picture is clarified. Local conditions which are unfavorable are: ocular or orbital inflammatory processes, turbidity of the transparent media, synechiae, aphakia and sclerotic processes of the retina and choroid. Likewise the following conditions signify a poor operative basis: absence of tears, excessive number or size of tears, presence of cicatricial bands in the vitreous, enormous extension of the detachment and greatly accentuated bulging of the retina at the level of the tears.

As general conditions that are unfavorable I mention deficient nutrition, tuberculosis, the dyscrasias and hepatorenal insufficiency. Patients with violent cough also represent poor risks. Those with chronic bronchial disorders and those suffering from prostatic involvement at times do not tolerate well a prolonged confinement to bed.

All these conditions contribute toward the determination of whether the condition is operable and aid in making the prognosis, for as I showed in my paper on the etiology of detachment of the retina, read before the International Congress of Ophthalmology at Madrid, Spain, the retina detaches when it is altered, but the alteration, which is at times local, is frequently accompanied by changes in the detoxifying organs (the liver and kidneys). In spite of all this, when one is confronted by the unfortunate visual future of a patient suffering from detachment of the retina, one must frequently operate even when conditions, both local and general, are not such as one would desire.

PREPARATION OF THE PATIENT

The preparation of the patient in a general way consists of: examination of the urine, subjection of the patient to a suitable regimen if necessary, and if he is a diabetic, rapid establishment of a normal content of sugar in the blood. If a hypertensive condition exists, this is likewise combated. If a cardiac, a hemophilic or a uremic condition is present, appropriate measures are taken according to the case. The intestinal tract is purged the night before operation, and no food is given the patient on the day of the operation. Other concurrent causes of the detachment (such as tuberculosis, syphilis or rheumatism)

are to be taken into consideration later, for in these cases it is a pity to lose time. Detachment of the retina, once established, is almost always a local condition, and its treatment, urgent and efficacious, is local. Investigation and treatment of the general condition can be made later.

The local preparation is more complicated. In the patient having a turbid vitreous as a result of recent hemorrhage, which interferes with a view of the retina, it is better to postpone the intervention until such time as the fundus is clarified, the patient being kept in relative repose during the period of waiting (he should not get out of bed very often and should wear a binocular bandage or stenopeic glasses). If the vitreous is turbid because of inflammatory exudate, it is better not to operate, for the turbidity does not clear readily.

When the state of the transparent media permits a view of the fundus, it is necessary to do everything possible to find the tear or tears and make note of their localization. To do this, it is essential to dilate the pupil and examine the fundus in its entirety. It is best to use a strong light in exploring the area, both direct and indirect methods being used. It is very useful to make a drawing of the fundus, even though it is roughly done, for it is important to have an exact reference to the position of the tears and the part detached.

It has been my practice for more than six years to note the situation of the tears, fixing the meridian in which they are located and the distance from the ora serrata. For the meridian I use the face of the clock; for the distance from the ora serrata I count the number of disk diameters by which the ora is separated from the center of the tear, and I multiply this number by 1½, which gives the approximate distance in millimeters. Since the limbus is about 8 mm. (from 9 to 10 mm. in patients with high myopia) anterior to the ora serrata, by adding the aforementioned figure to the latter the distance in millimeters from the limbus to the tear is obtained. Complicated apparatus for localization do not appear to be commendable.

INSTRUMENTARIUM

For the technic in which the galvanocautery is used, fine cautery points such as Vogt recommended (see figure 8 of his paper read in Madrid), or slightly thicker ones, are advised. With these, practically all detachments can be treated.

For diathermy it is immaterial whether the apparatus with a lamp or the one with a spark gap is employed. In general, all the apparatus on the market serve well. I utilize a diathermy machine made in Germany modified by Lacarrére, with complete satisfaction. For the electrode I use the "electrodiaphake" of Lacarrére, with a platinum or inoxidizable steel wire. The electrodes of Weve and Šáfár are useful and are the ones generally employed. The dose to be used cannot be

standardized; it varies between 100 and 150 ma., a series of factors, such as the electrode, the conductibility and the moisture on the area to which it is applied, making it very indefinite. It is better to be guided by the effects observed.

I do not use a pyrometric apparatus, i. e., an apparatus which measures the temperature at the point of action of the electrode, because the use of it is complicated and of slight benefit.

For trepanation, I use trephines from 1.5 to 2 mm. in diameter. As cauterizing solution, I employ 1 per cent caustic soda, and procure good results with it.

ANESTHESIA

Although cocaine may be instilled for anesthesia, I prefer 4 per cent *p*-aminobenzoyl-2, 2-dimethyl-3-diethyl-amino propanol hydrochloride because of its less damaging effect on the corneal epithelium. Instillations of epinephrine hydrochloride, 1:1,000, are alternated with the former, or the latter solution may be added to the anesthetic. While these instillations are being made, the meridian in which the tear or tears are located is determined with the aid of the ophthalmoscope. To do this, the patient is asked, after the tears are seen, to hold his vision fixed at a certain point. Then with a glass rod having a very fine tip, which has been moistened in an alcoholic solution of gentian violet (from 2 to 3 per cent), two colored points marking the site of the meridian are made at the limbus, this position being checked later with the ophthalmoscope.

After five or six instillations of the anesthetic, 0.5 cc. of 3 per cent procaine hydrochloride is injected into Tenon's capsule over the area of operation. Then several drops of the same solution are injected below the tendon of the inferior rectus and also beneath the skin of the lower lid. Finally an injection of 0.5 cc. of the solution is made retrobulbarly.

The injection below the rectus and that in the lid may be omitted if the patient is tractable and quiet. During the period of anesthetization the patient keeps his eyes closed, except at the moments of ophthalmoscopic examination.

The ordinary preparations for making the operative field aseptic are used.

OPERATION

The operation for detachment of the retina is not stereotyped like the extraction of a cataract or the trephining for glaucoma; it is more complicated in the sense that it is necessary to adapt the steps of the procedure to the individual case. Besides, there is a diversity of technical appliances, and because I have already published an article dealing with the most important of these, I shall limit myself here to a descrip-

tion of the most frequent type of operation and the different variations according to the clinical form of the detachment. I wish to state that the technics described here are only slightly original; they are the technics with minor variations adopted by the oculists who have devoted themselves especially to this class of intervention.

If the inferior rectus and the lower lid have been anesthetized, a curved needle carrying a rather thick silk suture thread is passed beneath the tendon or the muscle. Both ends of this suture thread are brought out through the lower lid, the points of exit on the surface of the skin being separated by from 5 to 6 mm. and being 3 or 4 mm. from the free border of the lid. At the same time they may be passed through a small celluloid plaque, over which they are loosely tied until the operation is completed.

A silk suture thread is now placed at the point on the limbus stained with gentian violet which is nearest the tear. The suture must be deep

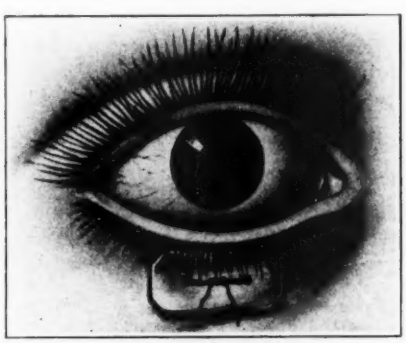


Fig. 1.—In unruly patients it is practical to pass a silk suture thread through the inferior rectus flush with the sclera, bringing it out through the lower lid and tying it over a celluloid plaque to prevent cutting of the skin.

enough to include the superficial layers of the sclera so that it will withstand the making of efficient traction. The suture may be dispensed with when the zone of intervention is anterior to the equator, especially if it is inferior or external, such a zone being the most accessible. A Kocher forceps is clamped on the ends of the suture thread.

An incision into the conjunctiva parallel with the limbus is made about 6 to 8 mm. from the cornea, using curved blunt-tipped scissors and cutting through the capsular tissues to the sclera. With the same scissors, Tenon's capsule is dissected off the sclera, the latter being left entirely bare. It is important in this step not to injure the vena vorticosae. If there are muscles in the region, the instrument is passed beneath them.

During these manipulations 1 per cent physiologic solution of sodium chloride is instilled on the cornea from time to time, or a contact lens (Weve) may be placed over the latter.

When the area has been denuded, the speculum is inserted. A model which I devised has proved very practical. Traction is now made on the suture thread fastened to the limbus, and a point on the sclera is stained with gentian violet to mark the extension of the meridian from the first markings. With a compass, the requisite number of millimeters is measured along this line as far as is necessary to encounter the area of the tear. This point also is designated by gentian violet stain or with india ink.

I shall describe a simple case in which there is a small tear at the level of the equator, with a flat detachment. If the thermocautery or galvanocautery is used, two small ignipunctures extending to a depth of from 2 to 3 mm. are made in the same area, permitting the escape of subretinal fluid. Immediately thereafter the area treated is examined with the ophthalmoscope (the operating room having been darkened), and a careful search is made for the punctures and the tear. If the thermopuncture is not seen (and frequently it is not, because one has not succeeded in penetrating the retina), the foregoing procedure is repeated in the same spot, the cautery being pushed a little deeper, after which one again examines the area with the ophthalmoscope. If in spite of this, the ignipuncture cannot be located, a small localization lance is introduced through the hole in the sclera, this plug being visible through the retina provided the cornea and vitreous are clear. The puncture appears as a whitish stain which at times has a dark central spot. If one has had the good fortune to strike the part right over the tear, and if the latter is small, the detachment may be cured with only this intervention. If the punctures were placed to the side of the tear, the procedure must be repeated, the new site being determined by calculating the distance in disk diameters between the tear and the punctures. Whatever is done must be controlled by ophthalmoscopic examination.

The ordinary thermocautery or galvanocautery may be used, but the fine galvanocautery points advised by Vogt have a decided advantage in that good drainage can be obtained with them, with minimal lesions, even though many punctures are made.

If diathermy is used, the same procedure is followed: first, a double puncture is made for orientation and then the punctures necessary for coagulating the zone of the tear. Only in those cases in which, in spite of the tear, the retina continues to be in contact with the choroid or very close to it may superficial diathermy instead of the perforating modality be utilized.

If the method of scleral trepanations followed by chemical irritation is used, exact localization is not quite so necessary, although a galvanopuncture or diathermopuncture of orientation may be made, or the orientation lance may be used, but because multiple trepanations are

EXPLANATION OF FIGURE 2

A, two points stained with an alcoholic solution of gentian violet mark the meridian in which the tear in the retina is located. One suture thread is used to draw the globe downward and inward; the other passes through the inferior rectus and out through the lower lid.

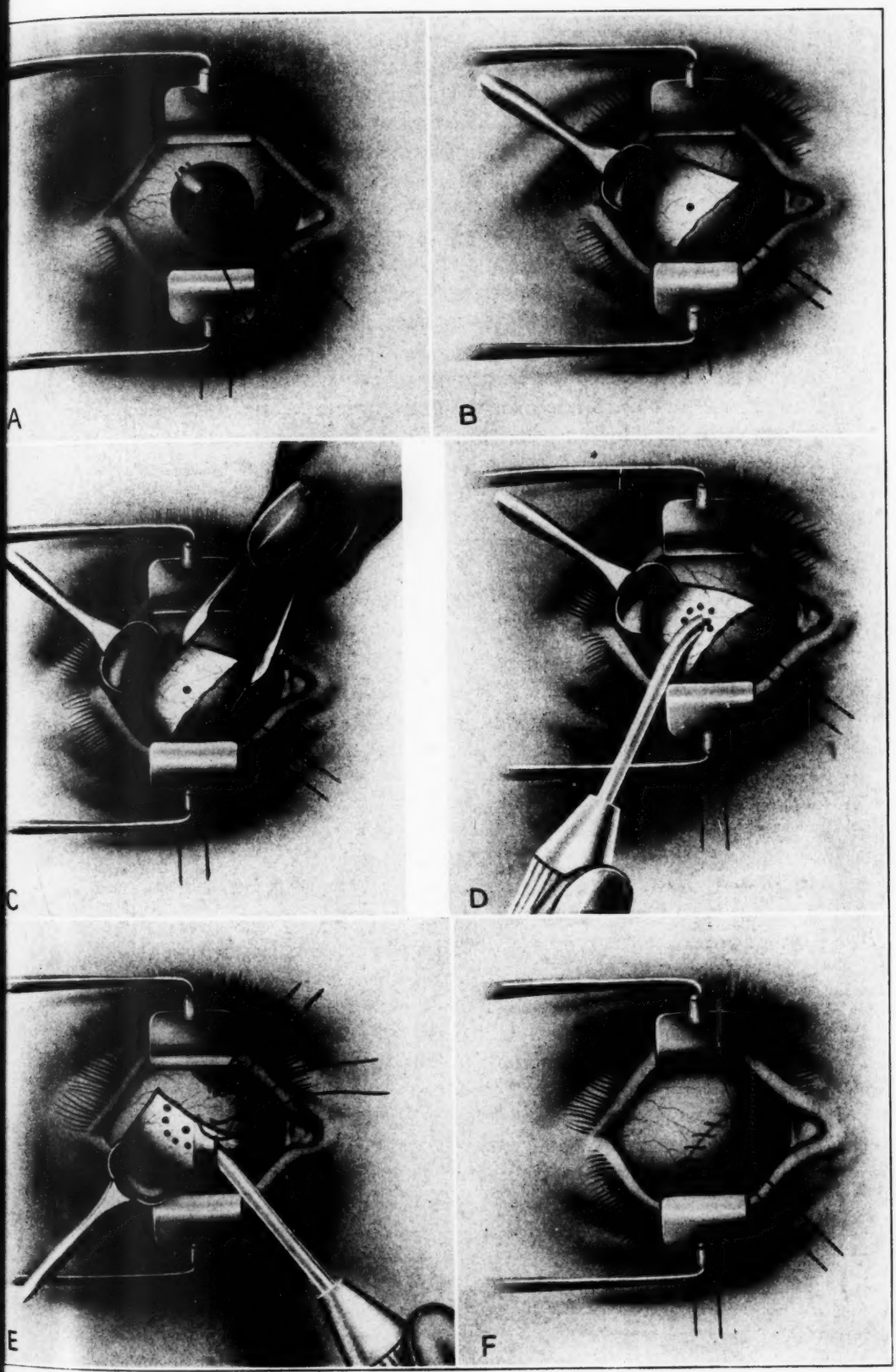
B, after the incision in the conjunctiva is made, the retractor shown in the illustration retracts Tenon's capsule and the orbital tissues, permitting the posterior part of the globe to be reached.

C, with a compass, the distance from the limbus is measured, the distance from the ora serrata to the retinal tear, which was computed ophthalmoscopically, being used in the calculation.

D, with a diathermy needle, or with the handle of Lacarrère, the sclera around the tear is perforated.

E, in this case the tear was a disinsertion in the ora serrata in the inferior external part, for which reason the silk suture thread in the inferior rectus was not passed through the lid. The handle of Lacarrère permits working under the muscle.

F, on termination of the operation, the conjunctiva is closed with a silk suture thread untied, which is very easily removed in eight days by simply pulling on one of the free ends.


A
B
C
D
E
F

made around the tear it is very improbable that this will not be included within the sector operated on. I do not insist on the detailed technic which I have published. In like manner I am not partial to the little used procedures in which carbon dioxide snow, irritating chemicals, etc., are employed.

If the tear is large and the thermocautery or galvanocautery is used, a wider cauterization or multiple cauterizations must be made, for it suits one's purpose to have some vitreous make its appearance externally which, adhering to the periocular tissues, acts as a retention plug holding the retina in contact with the borders of the openings. If diathermy is used, various punctures are made, surrounding the tear as closely as possible. This is also done when scleral trepanations are employed.

If the rents are multiple, the same technic is applied to each one. It is perfectly clear, of course, that after the eye is punctured, manipulation is more difficult, but with the cautery placed at reddish-white heat, or with the amperage of the diathermy augmented, the globe may be

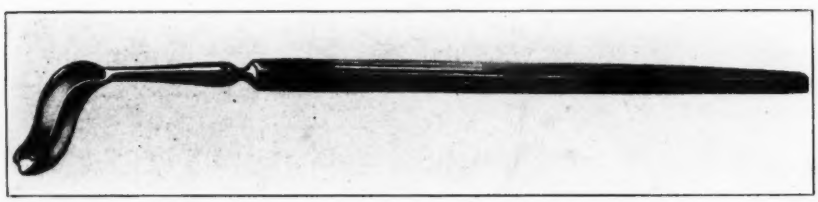


Fig. 3.—Retractor which I have had constructed for operating on detachment of the retina (sold by Mueller, Chicago).

perforated without making sufficient pressure to produce a further loss of vitreous. In the cases in which trepanations are utilized, the holes in the choroid can be closed with the small screws designed for this purpose.

However, the cauterizing action of the galvanic current and especially that of diathermy and chemical agents has a limit, and its use must be restricted as much as possible. It must be borne in mind that the subretinal fluid which does not escape during the operation, and that which is formed as a result of choroidal irritation and exudation after the intervention, following the closure of the scleral wounds, must be reabsorbed by the choroid if cure is to follow. The greater the reabsorptive power of the choroid the greater are the probabilities of a successful outcome. If the choroid is greatly irritated, or if it is atrophied, its absorptive powers are markedly decreased. Because of this, an old detachment of the retina is difficult to cure, for it is well known that with time the choroid of an eye attacked by detachment of the retina and not cured atrophies. I have observed many cases in which the therapeutic procedure had to be extensive in eyes that were in bad

condition, and in which intervention was followed by cyclitis and secondary glaucoma or by hemorrhage of the choroid.

In cases in which, because of multiplicity of tears or extension of the retinal lesions, it is necessary to resort to extensive intervention, it is better to do two or three operations, beginning with the upper tears. When the rents are located below the muscles, it is easy to work under my speculum, placed beneath the covering muscle. If the tears are far posterior, it is at times necessary to cut one of the muscles, although this step should be avoided if the other eye has good vision, because no matter how well one is able to sew the muscle in its original position, there frequently remains a molesting diplopia.

When the tear is in the macula, it is necessary to cut the external rectus, rotate the eye and apply the speculum, by which steps the optic nerve is easily approached. An area about 4 mm. this side of the entrance of the nerve into the globe is cauterized, and the effect obtained is observed with the ophthalmoscope.

When the retina reveals marked bulging at the level of the tears and consequently great separation from the choroid, the intervention will not be efficacious if the retina does not remain fastened to the latter after the operation, for to provoke an adhesive choroiditis serves no purpose if these two membranes do not remain in contact. In such cases, the use of the galvanocautery or that of trepanations is preferable to that of diathermy, for drainage must be ample. Diathermy may, however, be used, as good drainage as possible being procured. The escape of some vitreous externally contributes to the cure.

As an aid to keeping the retina fixed to the choroid I have practiced injection of air into the vitreous, making a perforation with the galvanocautery or diathermic needle 6 mm. from the limbus in the superior part subsequent to dissection of the conjunctiva. The syringe is filled with sterilized air aspirated during the time that the needle is at red heat. This needle, which is of fine caliber and of platinum, is introduced 2 or 3 mm. in the direction of the posterior pole, and from 1 to 2 cc. of the air is injected until the globe acquires a certain tension, which is visually determined. The procedure is well tolerated, but it is inconvenient in that it does not permit ophthalmoscopic control of the superior zone. From five to ten days are required for absorption of the air. I have used this method in nine cases only, and I cannot, therefore, as yet evaluate it in its entirety.

The tears in the ora serrata (disinsertions) must be treated by applying the therapeutic action, whatever the technic may be, over the border of the retina which surrounds them, forming the arc of a circle the extremities of which approach the ora serrata from the extremities of the tear. As is to be supposed in these cases, one or two applications of cautery or of diathermy are not sufficient; it is necessary to repeat the applications a number of times.

In the cases in which there are no tears or, to be more accurate, in which one does not see any tears, the results of intervention are problematic and frequently entirely negative.

Punctures of large sacs, even though they are used in conjunction with diathermy or trepanation with application of some caustic solution, and though they appear to produce improvement while the patient is absolutely quiet, are frequently followed by hopeless recurrences. At times the puncture may uncover a tear that previously was invisible, and during the intervention it is brought into view, which is a very favorable circumstance. Only in the cases in which the detachment is of a clinical type that permits a presupposition of the location of the tear can one attempt intervention where the tear is believed to exist, but this must always be stated with a question mark. In reality, the absence of a visible tear is a bad omen if the fundus is clear, as well as if the vitreous is turbid and a view of the retina difficult. In some cases, rest and repeated observation permit a determination of the procedure to follow.

When the intervention on the globe is completed, the limbal suture is removed and the conjunctiva is sewed (not Tenon's capsule) with a continuous silk suture thread untied, the ends of which extend 2 or 3 mm. beyond each extremity. This suture thread has the advantage of being removed easily and without pain by simply pulling one end of it with a forceps, following one instillation of an anesthetic, eight or ten days after the operation.

If because of nervousness the patient is unable to control himself and remain quiet, a retrobulbar injection of blood is made. From 8 to 10 cc. of blood is extracted from one of the veins of the arm, and this is injected, the needle being passed posteriorly through the inferior culdesac and externally to a depth of about 3 cm. This immobilizes the globe and so permits one to avoid bandaging the eye not operated on in those rare cases in which the binocular bandage cannot be tolerated. Ordinarily, after the instillation of atropine, a binocular bandage is applied, and the patient is put to bed.

POSTOPERATIVE PRECAUTIONS

I believe that rest of the globe is of much greater importance than rest of the body. Because of this, a certain amount of body movement is tolerated. In many cases, the patient is allowed to be in a semirecumbent posture. Position has only a relative value; the principal factor is rest of the eyes. It is my custom to place the patient on the side on which operation was performed.

If postoperative pain is intense I usually administer a product containing barbital and amidopyrine or acetyl salicylic acid. Ordinarily the pain is of short duration.

The day following operation a light diet, modified according to the tolerance of the patient, is prescribed. In three days the bandage is removed and atropine instilled. If rest in bed is poorly borne by the patient, he may now be allowed to get up. If the patient moves about too much, a retrobulbar injection of blood may be given, and this may be repeated every five or six days. In six days the bandage is again changed and atropine instilled. The fundus may now be examined with prudence, care being taken to avoid as much as possible all movement of the eye. Generally the eye not operated on is now left uncovered, the patient being counseled to keep it closed most of the time, or stenopeic glasses may be put on. If on examination of the fundus it is noted that the retina is still not adherent, or if it was known beforehand that treatment would be difficult, the binocular bandage is kept on several days more. The suture thread may be removed after the eighth day.

The bandage of the eye operated on is generally removed fifteen days after operation, while the stenopeic spectacles continue to be used and atropine is instilled every two or three days for a period of fifteen days more. During this time it is wise not to resort to violent exercise and, above all, not to incline the body forward.

COMPLICATIONS

On the part of the general state, various annoying conditions may supervene. Vomiting, however, does not constitute a complication to be feared as in the operation for cataract or glaucoma. Constipation is common, for which reason it is best to purge the bowels of the patient the night before operation. Hypostatic pneumonia may make its appearance in the aged patient. This is avoided by frequently changing the position. Dysuria occurring in the patient with a prostatic disorder at times requires catheterization of the bladder.

As to local complications, there may be, in the first place, failure of the retina to adhere, which is sometimes due to poor operative technic, in that the tears were not completely closed or isolated, and sometimes to the bad condition of the patient. This complication is generally manifested when the patient begins to walk and to move the eyes. *Therefore, one must not place too much faith in the apparent cure while the patient remains quiet.* The so-called recent recurrence, demonstrated by the presence of new tears, frequently represents an incomplete examination in the first place, the tears having existed at the time of the examination, but unsuspected or incapable of being seen.

When, after a complete reattachment and after it has been shown ophthalmoscopically that there are no tears, a new detachment with or without tears is produced, one knows that it is a true recurrence. This is frequently brought about by blows in the eye or on the head and by straining efforts, and at times, without apparent cause. It may also

be produced by retraction of the retina in the area surrounding the operative field. Such recurrences are not numerous; they occur to the extent of from 6 to 8 per cent. If the case is clear, reoperation must be done, although one must keep in mind that a large number of postoperative hemorrhages occur in cases in which operation is performed a second or a third time in a choroid hyperemic from former intervention.

With the exception of cases of the latter type, hemorrhage at the time of or after operation is not so frequent as it was at the time that operative treatment was first instituted, when thick cauteries were used and kept within the globe for a number of seconds (according to some authors, for as long as ten seconds). However, this grave complication which is so damaging to the patient, and which leaves the surgeon so helpless, has unfortunately not yet disappeared, although happily it is rare.

Uveitis is, in reality, a reaction of the uvea to the operative insult. If it is most marked in the ciliary body, it is very painful and capable of producing a hypertension or hypotension which predisposes the eye to glaucoma or atrophy. It is usually the result of extensive operation, although the result of such operation is influenced greatly by the condition of the "terrain," i. e., the previous state of the uvea, for there are times when widespread operations are well tolerated, while at other times even operations of moderate extent are not so well borne. Assuredly, if the choroid is the seat of an inflammatory process, the superposition of an adhesive choroiditis provoked with therapeutic intent will cause an exacerbation of the inflammation.

RESULTS

The results obtained have been progressively better for various reasons. In the first place, there is today much greater facility in the separation of cases which are operable and those which are not. Second, the operative procedures, as well as the instruments used, are more perfected. In the third place, there are available in the literature the data which the experience of the various ophthalmologists who practice operative treatment of detachment of the retina has produced. Finally, experience has improved technic and has permitted a more effective avoidance of the operative difficulties, which in these interventions are primarily numerous.

In the statistics which were published at the Congress of Ophthalmologists at Amsterdam, the Netherlands, in 1929, it was shown that in 33 cases 11 cures had been obtained. In the congress at Santander, Spain, about two years ago, it was stated that 164 patients had been operated on, with 53 cures. From June 1, 1932, to Aug. 1, 1934, 248 patients suffering from detachment of the retina were operated on and reattachment was obtained in 168, but since 27 of

these patients had, in spite of the anatomic cure, vision of less than 0.15 because of preexisting lesions or because of the age of the detachment, there remained 141 patients with vision better than 0.15, or more than half of the series with good results.

I hope that these figures will be improved as time goes on. It is very probable that they will be, because all oculists are operating on their patients earlier, and the patients in turn are submitting more readily to operation.

These results should serve to stimulate all my colleagues in this work to persevere in the use of such treatment. Even if the percentage of good results is not large in the beginning, this is no reason why the course on which we are embarked should not continue to be followed. From the humanitarian point of view it is preferable to have many oculists operate, obtaining 30 per cent of cures, rather than a few, with 55 per cent of cures, for with time the former will also obtain the higher percentage of good results, and probably more.

CONCLUSIONS

Operative treatment of detachment of the retina constitutes the means of restoring vision to more than half of the patients suffering from this disease.

Its success is based on: prompt execution, search for retinal tears, their localization and their obstruction or isolation by means of operative intervention.

The choice of operative method, except in special cases, is secondary, for the same effects can be obtained with thermocautery, galvanocautery, diathermy or trepanations. In general, however, it may be said that diathermy is the method having the greatest advantages. It is indispensable to have a good localization preoperatively and to have ophthalmoscopic control of the various steps during the procedure. It is a question, as I stated in the congress at Heidelberg, Germany, of *much patience in the dark room and much patience in the operating room.*

OBSERVATIONS ON FOUR THOUSAND OPTIC FORAMINA IN HUMAN SKULLS OF KNOWN ORIGIN

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This communication presents certain phases of an investigation, conducted by the courtesy of Dr. T. Wingate Todd, on the optic foramen and its contiguity as represented in the skulls housed in the Hamann Museum of the Laboratory of Anatomy at Western Reserve University.

MATERIAL

The material which formed the basis of this study consisted of 2,187 skulls. Of these 123 were wet (preserved in fluid); the remainder were dried. According to race and sex, 1,354 were from white persons (1,146 from males and 208 from females) and 833 were from Negroes (599 from males and 234 from females). The ages of the subjects ranged from 1 day to a stated age of 105 years.

The skulls were prepared for research by being sectioned in the sagittal plane, or the calvarium was removed at necropsy. It was possible to inspect minutely their interiors. The age, race and sex of the subjects and in most instances the cause of death are recorded.

For many years anatomists have known that in some persons there are, immediately adjoining the optic foramen posteriorly, two osseous canals (fig. 2). The more anterior of these canals (the clinocarotid) is traversed by the internal carotid artery; the more posterior (the interclinoid), by a communication between the circular and the cavernous venous sinus. In other persons both the artery and the venous sinus are found in the same foramen, the common interclinoid (fig. 5). Also, the floor of the optic foramen occasionally bears a groove or, more rarely, a foramen capable of housing the ophthalmic artery. It is proposed to describe these variations as found in the collection of skulls preserved at the Western Reserve University.

THE CLINOCAROTID CANAL (ANTERIOR INTERCLINOID CANAL)

The clinocarotid canal is formed by a union of the anterior clinoid process on its mesial side with the tip of the middle clinoid process as it arises from the tuberculum sellae or the lateral wall of the body of the sphenoid bone. The osseous boundaries of the canal are: laterally,

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the anterior clinoid process; posteriorly, a narrow bridge of bone joining the anterior and middle clinoid processes; mesially, the body of the sphenoid bone where it bears the anterior end of the sulcus caroticus; anteriorly, and occasionally as a partial roof, the posterior root of the lesser wing of the sphenoid where it forms the floor of the optic foramen.

It is this proximity of the internal carotid artery to the optic nerve as it enters its foramen that is of particular interest to clinicians. Because of this interest 2,187 skulls were inspected, and the frequency of this anomaly was recorded.

A clinocarotid canal in some form was found in 762 skulls (34.84 per cent); the structure was bilateral in 512 and unilateral in the remainder, being found on the right side in 142 and on the left in 108. A more detailed analysis is given in table 1.

TABLE 1.—*Classification of 762 Skulls (34.84 Per Cent) which Contained Some Form of Clinocarotid Canal* *

	Bilateral		Unilateral (Right)		Unilateral (Left)	
Type of Canal	No. of Skulls	Per-centage	No. of Skulls	Per-centage	No. of Skulls	Per-centage
Complete	117	5.35	20	0.91	18	0.82
Contact	7	0.32	13	0.59	7	0.32
Incomplete	227	10.37	109	4.98	83	3.80
Mixed	161	7.36				
Total	512	23.41	142	6.49	108	4.93

* The percentage is figured on a basis of 2,187 skulls.

The racial distinction was interesting. A clinocarotid canal was observed in 40.91 per cent of the skulls of white persons and in 24.96 per cent of those of Negroes, a variation of 5:3. The frequency in white males surpassed that in Negro males by 15 per cent. For the females of both races the differentiation was 16 per cent. Differentiation as to sex was not marked in skulls from persons of the same race. The exact percentages are given in table 2.

The ages of the subjects in whose skulls a clinocarotid canal was present ranged from 21 days to a stated age of 105 years. Completely ossified canals were noted in skulls of children of the following ages: 21 and 30 days and 3, 7 and 15 months.

Investigation revealed three types of clinocarotid canal:

1. The complete type in which a bony ring or foramen was present (fig. 2).

2. The contact type, in which a foramen was apparently formed, but in which close inspection of the posterior wall revealed a dividing line or suture (fig. 3).

3. The incomplete type, which showed the middle clinoid process and a spicule of bone extending mesially from the anterior clinoid

process, but in which a gap existed in the posterior wall of the canal (fig. 7).

INTERCLINOID OSSEOUS BRIDGE, COMMON INTERCLINOID CANAL AND POSTERIOR INTERCLINOID CANAL

A knowledge of the incidence of an osseous interclinoid bridge and its recognition are of increasing importance as the significance of changes in the suprasellar region becomes better known.

TABLE 2.—*Classification According to Race and Sex of 762 Skulls Containing Some Form of Clinocarotid Canal* *

Type of Canal	White Males		White Females		Total White Persons		Negro Males		Negro Females		Total Negroes	
	Number of Skulls	Percentage	Number of Skulls	Percentage	Number of Skulls	Percentage	Number of Skulls	Percentage	Number of Skulls	Percentage	Number of Skulls	Percentage
Bilateral												
Complete	59	5.14	10	4.80	69	5.09	34	5.67	14	5.98	48	5.76
Contact	4	0.34	2	0.96	6	0.44	1	0.16	0	....	1	0.12
Incomplete	161	14.04	24	11.53	185	13.66	29	4.84	13	5.55	42	5.04
Mixed	96	8.37	18	8.65	114	8.41	37	6.17	10	4.27	47	5.64
Unilateral (right)												
Complete	10	0.87	2	0.96	12	0.88	6	1.00	2	0.85	8	0.96
Contact	6	0.52	0	....	6	0.44	7	1.16	0	....	7	0.84
Incomplete	74	6.45	14	6.73	88	6.49	15	2.50	6	2.56	21	2.52
Unilateral (left)												
Complete	9	0.78	1	0.48	10	0.73	6	1.00	2	0.85	8	0.96
Contact	6	0.52	0	....	6	0.44	1	0.16	0	....	1	0.12
Incomplete	48	4.18	10	4.80	58	4.28	20	3.33	5	2.13	25	3.00
Total	473	41.27	81	38.94	554	40.91	156	26.04	52	22.22	208	24.96

* The percentage is calculated in terms of the total number of skulls from white persons, 1,354 (1,146 from males and 208 from females), and of the total number of skulls from Negroes, 833 (599 from males and 234 from females).

TABLE 3.—*Classification of 190 Skulls (8.68 Per Cent) which Contained Some Form of Osseous Interclinoid Bridge Attached to the Posterior Clinoid Process* *

Type of Bridge	Bilateral		Unilateral (Right)		Unilateral (Left)	
	No. of Skulls	Percentage	No. of Skulls	Percentage	No. of Skulls	Percentage
Complete	41	1.87	23	1.05	34	1.55
Incomplete	24	1.09	27	1.23	28	1.28
Mixed	13	0.59				
Total	78	3.56	50	2.28	62	2.83

* The percentage is figured on a basis of 2,187 skulls.

In the series of 2,187 skulls some form of interclinoid osseous bridge attached to the posterior clinoid process was found in 190 specimens (8.68 per cent). As in the case of the clinocarotid canal, the osseous bridges were complete or incomplete, but no contact bridges were noted. Table 3 gives an analysis according to the type and distribution.

These bridges were unilateral or bilateral. When present bilaterally they limited the hypophyseal fossa laterally (fig. 2). They connected the posterior, middle and anterior clinoid processes, the posterior and anterior processes or, rarely, the posterior and middle processes. Frequently the union was not completed, and spicules of bone projected from the clinoid processes (fig. 1).

When an interclinoid osseous bridge is present and the clinocarotid canal is absent, a large foramen is formed which transmits the internal carotid artery anteriorly and the lateral part of the circular sinus posteriorly. This foramen was named by Gruber [1] the common interclinoid foramen. It is bounded ventrally by the body of the sphenoid, dorsally by the interclinoid bridge, anteriorly by the posterior root of the lesser wing of the sphenoid (floor of the optic foramen) and pos-

Fig. 1 (skull 1,441)).—A dorsal view of the body and lesser wings of the os sphenoidale. *1* indicates the cranial aperture of the optic foramen; *2*, the sulcus caroticus; *3*, the right anterior clinoid process, showing an incomplete bridge; *4*, the left anterior and posterior clinoid processes, joined by an osseous bridge, and *5*, the dorsum sellae, bearing the posterior clinoid processes. The middle clinoid processes are absent.

teriorly by the posterior clinoid process as it joins the dorsum sellae. When a clinocarotid (anterior interclinoid) canal, whether complete or incomplete, exists, this common foramen is divided anteriorly by a complete or incomplete bony partition which consists of the posterior wall of the clinocarotid canal. This division forms a small anterior interclinoid (clinocarotid) arterial canal and a larger posterior interclinoid venous canal. A common interclinoid foramen (no evidence of a clinocarotid canal) was present in 19 skulls (0.86 per cent). There was fusion of the middle and anterior clinoid processes in 155 craniums (272

1. Gruber, W. L.: Beiträge zur Anatomie des Keilbeins und Schläfenbeins, St. Petersburg, Eggers u. Comp., 1859.

instances). In 98 skulls (139 instances) the posterior clinoid process was united with the anterior process by an osseous bridge (figs. 2 and 5).

This is at variance with the observations of some observers as quoted by Le Double.[2] "According to Meckel, Hyrtl, Cruveilhier, Calori, etc., when the middle clinoid apophysis is fused with the anterior it is always also with the posterior."

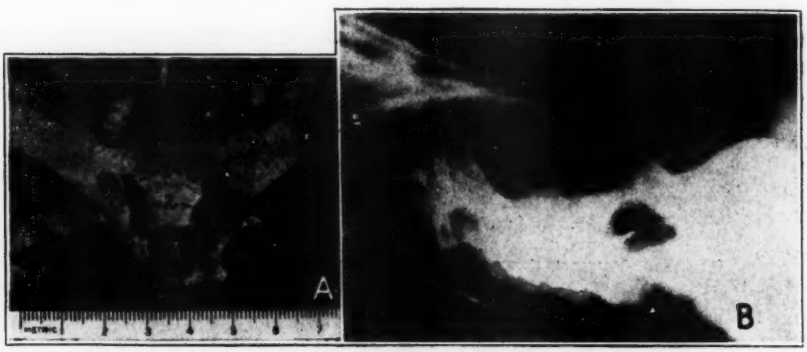

Fig. 2 (skull 1,651).—In *A*, all three clinoid processes are united bilaterally by osseous bridges. *1* indicates the optic foramen; *2*, a complete clinocarotid or anterior interclinoid canal or foramen, and *3*, the hypophyseal fossa framed by bridged clinoids. The posterior interclinoid foramen is hidden by its roof—the united posterior and anterior clinoid processes. *B* is a left lateral roentgenogram of the sella turcica.

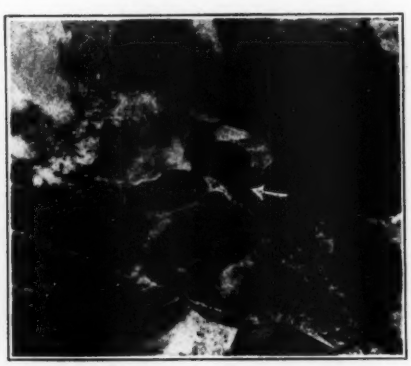

Fig. 3 (skull 354).—A right clinocarotid canal of the contact type. Note the suture line in the posterior wall as indicated by the arrow.

An analysis of the distribution of the interclinoid bridge on the basis of race and sex showed a greater frequency in skulls from Negroes, the skulls from females excepted. This was a mild reversal of the situ-

2. Le Double, A. F.: Traitè des variations des os du crâne de l'homme et de leur signification au point de vue de l'anthropologie zoologique, Paris, Vigot Frères, 1903, p. 273.

ation noted for the occurrence of the clinocarotid canal. Details are recorded in table 4. The youngest subject whose skull contained a bridge was a white boy aged 6 years. The oldest was a person 80 years old.

Fig. 4.—A dissection showing a complete clinocarotid canal in the skull of a Negro boy aged 7 months. *1* indicates the optic nerve, sectioned immediately posterior to the cranial opening of the optic foramen; *2*, the ophthalmic artery as it arises from the internal carotid artery, *3*, and passes under the optic nerve to the optic foramen; *4*, the posterior wall of a clinocarotid osseous canal. The soft parts are held forward by a probe.

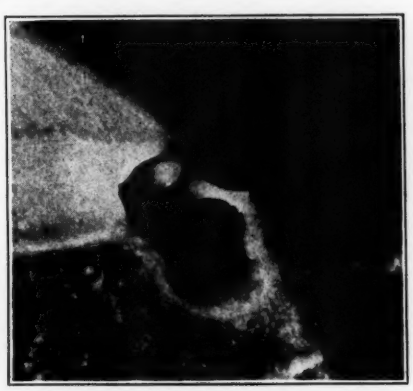

Fig. 5 (skull 1,784).—A lateral view of a common interclinoid canal or foramen formed by the union of the right posterior and anterior clinoid processes. The middle clinoid process is absent.

Two rare conditions were found, each being present only once in the 2,187 craniums. The first was found in skull 2,009, that of a Negro,

aged 30 years. It consisted of a partition of bone which on the left side joined the posterior and middle clinoid processes with the body of the sphenoid. Dorsal to the middle clinoid process was a groove 6.5 mm. wide by 3.5 mm. deep, apparently to accommodate the venous vessels in the absence of a foramen. The anterior and middle clinoid processes were bridged, forming a left clinocarotid canal. On the right side a clinocarotid canal and a very small venous or posterior interclinoid foramen were present. There was a shallow notch in the interclinoid bridge in the same location as the groove on the left side (fig. 6).

The second rarity was in skull 1,319, that of a Negro, aged 39 years. The left posterior and middle clinoid processes were bridged, but a gap

TABLE 4.—*Classification According to Race and Sex of 190 Skulls Containing Some Form of Osseous Interclinoid Bridge Attached to the Posterior Clinoid Process* *

	White Males		White Females		Total White Persons		Negro Males		Negro Females		Total Negroes	
Type of Bridge	Number of Skulls	Percentage	Number of Skulls	Percentage	Number of Skulls	Percentage	Number of Skulls	Percentage	Number of Skulls	Percentage	Number of Skulls	Percentage
Bilateral												
Complete	14	1.22	1	0.48	15	1.10	22	3.67	4	1.70	26	3.12
Incomplete	8	0.69	3	1.44	11	0.81	9	1.50	4	1.70	13	1.56
Mixed	3	0.26	2	0.96	5	0.36	6	1.00	2	0.85	8	0.96
Unilateral (right)												
Complete	12	1.04	3	1.44	15	1.10	8	1.33	0	....	8	0.96
Incomplete	11	0.95	5	2.40	16	1.18	10	1.66	1	0.42	11	1.32
Unilateral (left)												
Complete	13	1.13	3	1.44	16	1.18	13	2.17	5	2.13	18	2.16
Incomplete	18	1.57	3	1.44	21	1.55	5	0.83	2	0.85	7	0.84
Total	79	6.89	20	9.61	99	7.31	73	12.18	18	7.69	91	10.92

* The percentage is calculated in terms of the total number of skulls from white persons, 1,354 (1,146 from males and 208 from females), and of the total number of skulls from Negroes, 833 (599 from males and 234 from females).

existed between the middle and anterior processes, forming an incomplete bilateral carotid canal (fig. 7).

FORAMINA AND SULCI IN THE FLOOR OF THE OPTIC FORAMEN

It is not unusual to find the lesser wing of the sphenoid, as it forms the floor of the optic foramen, pierced by a tunnel. On rare occasions this foramen is sufficiently large to contain the ophthalmic artery. In 1 cranium, skull 2,404, that of a Negro female aged 60 years, this condition was discovered at dissection. The left ophthalmic artery traversed a foramen measuring 2.4 mm. by 2.6 mm., while the right was found in a partly closed groove measuring 2.4 mm. by 3.4 mm. (fig. 8).

For statistical purposes a foramen with a minimum diameter of 2 mm. was considered capable of containing an ophthalmic artery or a branch. Among the 2,187 skulls inspected, such a foramen was found

in 5—only 0.22 per cent. An analysis of these craniums failed to reveal a bilateral occurrence, the nearest approach being in skull 2,404, already described. The condition was found unilaterally on the right side in the skull of a Negro female and on the left side in skulls from 1 white male, 1 Negro female and 2 Negro males.

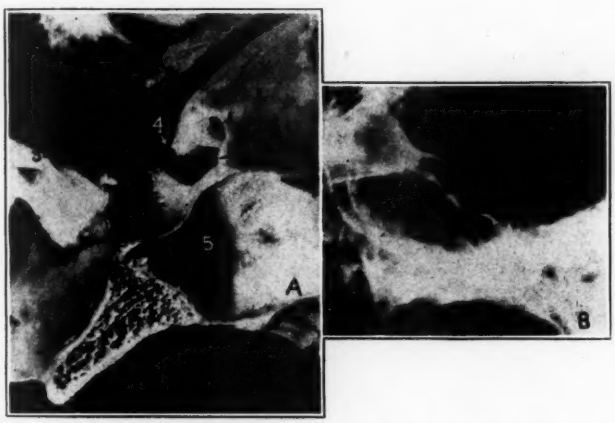

Fig. 6 (left half of skull 2,009).—The part of the skull shown in *A* presents a rare condition: A thin partition or wall of bone unites the posterior and middle clinoid processes with the body of the sphenoid bone. *1* indicates the optic foramen; *2*, a clinocarotid canal; *3*, united posterior and anterior clinoid processes; *4*, a notch above the middle clinoid process, and *5*, the sinus sphenoidalis. *B* is a left lateral roentgenogram of the sella turcica.

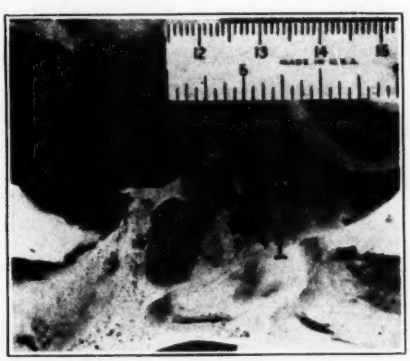

Fig. 7 (skull 1,319).—This part of the skull presents a rare condition. The left posterior and middle clinoid processes are united, forming a posterior interclinoid canal, indicated by the arrow. The anterior clinoid process and a spicule of bone from the middle process change the sulcus caroticus into an incomplete clinocarotid canal (1).

More frequently a sulcus or groove was observed in the floor or at the junction of the floor and the outer wall of the optic foramen. In each instance it was partly separated from the lumen of the foramen

by spicules of bone and was sufficiently large to have contained an ophthalmic artery. Such grooves were present in 36 skulls, or 1.64 per cent of the craniums examined.

Only 1 skull contained a groove on one side and an ophthalmic foramen on the other. In 11 craniums bilateral sulci were observed. A sulcus was found unilaterally on the right side 9 times and on the left 16 times. The 11 bilateral instances were distributed according to race and sex as follows: Three occurred in skulls from white persons (1 male and 2 females), and 8 were found in skulls from Negroes (5 males and 3 females). The unilateral sulci were found in 8 skulls from white persons (7 males and 1 female) and 17 from Negroes (12 males and 5 females). The skulls of 833 Negroes were inspected as against those of 1,354 white persons; therefore, the preponderance in Negroes was even greater than appeared.

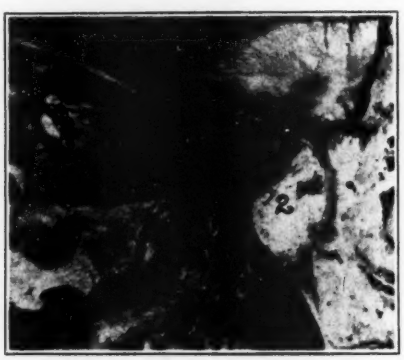

Fig. 8 (skull 2,404).—A view showing the posterior orifice of the left optic foramen (*1*) and the relationship of a second foramen (*2*) which was traversed by the left ophthalmic artery.

RELATIONSHIP OF FOREGOING ABERRATIONS AND ROENTGEN DELINEATION

Roentgenograms were made of the optic foramina in the skulls showing the foregoing anomalies to ascertain their relationship to the foramina. It was found that the clinocarotid canal did not encroach on the lumen of the foramen in properly taken roentgenograms. The larger foramina in the floor of the optic foramen can be seen roentgenographically; the sulci can be detected roentgenographically more easily, provided the central pencil of the roentgen rays is alined, or nearly so, with the axis of the optic foramen and the shadow does not fall into that of the orbital margin, obscuring details (fig. 20). A postero-anterior interclinoid bridge can best be seen in a lateral view of the sella turcica (fig. 2 *B*).

THE PROJECTED OPTIC FORAMEN AT THE PLANE OF THE ADITUS ORBITAE

The delineation by roentgenography of the optic foramen has shown vast improvement since the researches of Winckler [3] in 1901 and since Rhese [4] in 1911 called attention to its outline in the orbit with the ethmoid cells when a roentgenogram of the head was taken in the oblique position. From a survey of the literature on the subject it was gathered that unless the shadowy outline of the canal appeared near the inferolateral margin in the lower outer quadrant of the orbit, as depicted on the roentgenogram, the technic was incorrect (Goalwin,[5] Pfahler,[6] White,[7] Pfeiffer [8] and Giordano [9]). A few quotations will elucidate the point.

Whatever the direction of the axis of the optic canal may be in the normal individual of a given race and age, its continuation invariably strikes the lower outer angle of the margin of the orbit. It is, therefore, there that we should expect to find it in a properly taken optic canal roentgenogram (Goalwin [5d]).

. . . a knowledge of the direction of the axis of the optic canal is essential in the study of the shape and size of its cross section. In normal adults this axis is directed downward and outward toward the lower outer angle of the orbital margin (Goalwin [5e]).

. . . we should keep in mind that in all instances this canal must be projected near the lower outer border of the rim of the orbit, and unless it is found there, it is better to ignore the observations with regard to this canal (Pfahler [6]).

The image of the canal should always appear in the lower outer quadrant of the orbit (White [7]).

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3. Winckler, Ernst: Die Orientierung auf dem Röntgenbilde des Gesichtsschädels und das Studium der oberen nasalen Nebenhöhlen auf demselben, Fortschr. a. d. Geb. d. Röntgenstrahlen **5**:147, 1901.

4. Rhese: Die chronischen Entzündungen der Siebbeinzellen und der Keilbeinhöhle, Arch. f. Laryng. u. Rhin. **24**:384, 1911.

5. (*a*) Goalwin, H. A., and Schüller, A.: Die anatomischen und geometrischen Grundlagen für die röntgenologische Untersuchung des Canalis opticus, Ztschr. f. Augenh. **54**:103, 1924. (*b*) Goalwin, H. A.: Die exakte radiographische Darstellung des Canalis opticus, Fortschr. a. d. Geb. d. Röntgenstrahlen **32**:218, 1924; (*c*) The Precise Roentgenography and Measurement of the Optic Canal, Am. J. Roentgenol. **13**:480 (May) 1925; (*d*) The Clinical Value of Optic Canal Roentgenograms, Arch. Ophth. **60**:1, 1926; (*e*) One Thousand Optic Canals: Clinical, Anatomic and Roentgenologic Study, J. A. M. A. **89**:1745 (Nov. 18) 1927.

6. Pfahler, G. E., in discussion on Goalwin.[5f]

7. White, Leon E.: Anatomic and X-Ray Study of the Optic Canal in Cases of Optic Nerve Involvement, Ann. Otol., Rhin. & Laryng. **33**:121 (March) 1924.

8. Pfeiffer, Raymond L.: A New Technique for Roentgenography of the Optic Canals, Am. J. Roentgenol. **29**:410 (March) 1933.

9. Giordano, Giovanni: Ulteriore contributo alla tecnica ed all'anatomica radiografica del canale ottico, Riv. di radiol. e fis. med. **6**:803, 1931.

There is a latitude of projection in the lower outer quadrant of the orbit which will show the wall of the canal in accurate contour. The proper projection of the canal is in the center of this area (Pfeiffer [8]).

Prolonged toward the front the optical axes come to fall very near the infero-external angle of the orbit, touching at times its lateral wall (Giordano [9]).

When it was found in the roentgenograms that the outlines of many of the foramina did not appear near the inferior margin in the lower outer orbital quadrant, an investigation of the relationship of the projected axis of the canal and the plane of the orbital rim or aperture was undertaken. For this purpose an instrument that did not closely fill the foramen, such as a probe, was found inaccurate. Therefore, a drilled metal cone was devised which snugly fitted the foramen. Through its long axis was extended a movable metal rod which indicated anteriorly and posteriorly the long axis of the canal. By reading the point of contact on a quadrant placed over the orbit the exact projection of the axis anteriorly at this plane was ascertained. Also, by extending the rod to the calvarium the point of incidence of the same axis was again indicated.

By this means it was possible to ascertain where the outline of the foramen should appear in relationship to that of the orbit and also where the central pencil of roentgen rays should enter the skull to traverse the long axis when properly alined and to delineate the optic foramen accurately.

The relationship between the projected long axis of the optic foramen and the plane of the orbital rim or entrance (aditus orbitae) was studied in 1,000 foramina, in 500 skulls. One half of these craniums were from white persons and one half were from Negroes; according to sex, 125 were from males and 125 from females, white and Negro. The results are shown in figure 9.

It is worthy of note that the axis was not projected into the mesial half of any orbit. It extended from the midline of the orbit to behind the outer rim and from high up in the upper outer orbital quadrant to behind the body of the zygomatic bone.

The greatest incidence was centered along the horizontal midline at the junction of the upper and lower outer orbital quadrants (line *A-B*, fig. 9). Within a distance of 5 mm. above and 5 mm. below this line 322 axes were found in the right orbit and 350 in the left orbit, a total of 672, or 67.2 per cent. When this area was extended 5 mm., that is, made to include the space enclosed by a rectangle 20 mm. in the vertical by 30 mm. in the horizontal dimension, bisected horizontally by the midline (line *A-B*, fig. 9), an incidence of 450 axes was counted in the right orbit and of 461 axes in the left orbit, giving a total of 911, or 91.1 per cent.

Point *B* in figure 9 represents the center of any orbit and is a constant. Points *A* and *B* are 30 mm. apart. Point *O* is midway. The greatest concentration of axes fell in the four squares surrounding point *O*. Two hundred and twenty-eight axes were observed in the right orbit and 240 in the left, or a total of 46.8 per cent. Eighty-two and four-tenths per cent of the total number of axes were found in a rectangle (in bold outline) measuring 15 mm. vertically by 20 mm. horizontally and extending from 5 mm. above line *A-B* to 10 mm. below and to within 5 mm. of points *A* and *B*, respectively, or 10 mm. on either side of point *O*. This zone in the right orbit contained 83.4 per cent of the projected axes, and in the left, 81 per cent. In 84 instances (8.4 per cent) the axis was tangent to the orbital rim. In 126 (12.6 per cent) it projected through the bony orbital margin, leaving it within the outer half of the orbital aperture in 79 per cent.

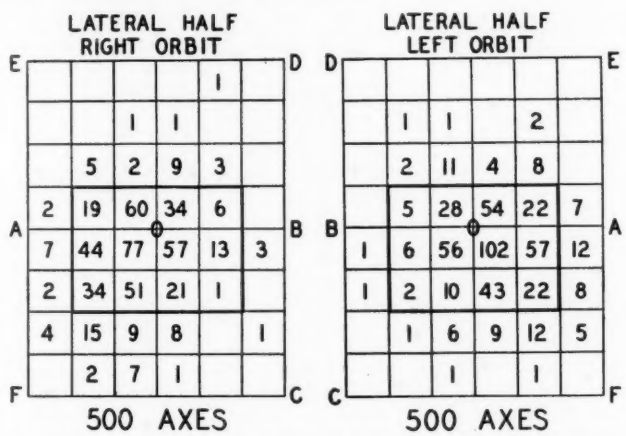

Fig. 9.—A graphic record of the projection at the plane of the aditus orbitae of 1,000 optic foramina. Line *A-B* represents the horizontal midline of the orbit (30 mm.); line *C-D*, the vertical midline of the orbit; *B*, the center of the orbit; *A*, a point beyond the lateral or temporal orbital rim; *O*, the midpoint of line *A-B*. Each small square equals 5 mm. by 5 mm.

An analysis according to race and sex of the incidence of axes in the outer half of the orbit, that is, not tangent to or behind the orbital margin, revealed the following percentages for the 500 subjects whose skulls were studied: according to race—in white persons, 74.4 per cent, and in Negroes, 83.6 per cent; according to race and sex—in white males, 78.4 per cent, in white females, 70.4 per cent, in Negro males, 87.2 per cent, and in Negro females, 80 per cent; according to sex—in males, 82.8 per cent, and in females, 75.2 per cent.

The racial differentiation was not noteworthy in the matter of tangency to the orbital margin. The incidence was 44 and 40, respectively,

in 500 axes observed in white persons and 500 observed in skulls of Negroes.

In the frequency of projection of the axes through the bony rim of the orbit there was a marked preponderance in the skulls of white persons. In this series 84 such projections were found in the skulls of white persons and 42 in those of Negroes. This made a ratio of 2 to 1 in favor of the white race.

A differentiation as to sex also existed. Tangency of the axis to the orbital rim predominated in skulls from females. The condition was found in 18 axes in skulls from male and in 26 axes in skulls from female white persons. For the Negro race the incidence was 15 for males and 25 for females, respectively.

Projection through the orbital rim was more frequent in the skulls of females of both races than in the skulls from males. This was demonstrated when it was found that in the respective samples of 500 axes the incidence was as follows: In 36 instances in skulls from white males and in 48 in skulls from white females and in 17 instances in skulls from male and in 25 in skulls from female Negroes this axis passed through the orbital margin.

In the light of the foregoing observations the more or less settled belief that the anteriorly projected foraminal axis normally falls near or through the lower lateral orbital margin must be modified.

Apparently Lysholm [10] realized this, and Frimann-Dahl [11] subscribed to his view when he quoted Lysholm as follows: " 'The optic foramen should' if possible 'appear near the middle of the orbit.' " The insert "if possible" was made by Frimann-Dahl to express the difficulty he experienced in controlling the location of the shadow of the optic foramen in the orbit.

This difficulty is not hard to explain if one considers that in 500 skulls no optic foramen was projected into the mesial (nasal) half of the orbit; that 28.4 per cent of the foramina were found in or lateral to the upper, outer (lateral) orbital quadrant and the remaining 71.6 per cent in or beyond the lower outer quadrant of the orbit. Moreover, asymmetry was the rule and not the exception. In 80 skulls (16 per cent of the sample) the optic foramina were symmetrical to within 5 mm. In 84 per cent (420 craniums) the axes were separated by 5 mm. or more when projected on the orbital quadrant (fig. 10). Symmetrical foramina were found in 13.8 per cent of the skulls from the white and in 17 per cent of those from the Negro group; in 17 per cent of the skulls of males and in 14 per cent of those of females;

10. Lysholm, E.: Apparatus and Technique for Röntgen Examination of the Skull, Acta radiol., supp. 12, 1931, p. 1.

11. Frimann-Dahl, J.: A New Projection for the Roentgenology of the Optic Foramen, Acta radiol. **14**:470 (July) 1933.

in 16.8 per cent of the skulls of white males, in 12.8 per cent of those of white females, in 19.2 per cent of those of Negro males and in 15.2 per cent of those of Negro females.

ANGULATION OF SKULL AND ROENTGENOGRAPHY

Interest was then centered on how much the skull could be tilted and still show an accurate delineation of an optic foramen. Experimentation with tilting the skull while the roentgen tube remained at right angles to the film demonstrated that a 5 degree tilt of the long axis of the optic foramen produced a slight distortion, while a 10 degree tilt caused displacement, inclusions and inaccuracy.

Fig. 10.—A view showing an orbital quadrant in position over an orbit and a semidiagrammatic representation of the area at the plane of the orbital entrance occupied by 82.4 per cent of anteriorly projected foraminal axes.

ROENTGENOGRAPHY OF THE OPTIC FORAMEN

This experience led to an investigation of several roentgenographic methods of depicting the optic foramen. Skulls were used with optic foramina the projected axes of which passed through the plane of the orbital aperture near the center, at extremes and at intermediate points. Skulls having the following anomalies were also used: a clinocarotid canal, a common interclinoid foramen, an ophthalmic foramen, a groove in the floor of the optic foramen and an optic foramen with the floor missing.

The methods of Goalwin,[11a] Camp and Gianturco [12] and Pfeiffer [8] were employed, as inquiry elicited the information that their procedures, or modifications thereof, were in general use in most of the larger medical centers of the United States. The originators [13] of two of the technics were interviewed.

The Goalwin method is an example of a technic in which all the maneuvering for the postero-antero-oblique position is made by angulation of the patient's head. The roentgen tube and film remain in a constant position and at right angles to each other.

The procedure of Camp varies from that of Goalwin in its simplicity. With a pronged metal localizer one endeavors to place the head in the proper oblique position "without the necessity of measuring certain angles or using special head rests."

The technic of Pfeiffer is an example of crossed projection. It permits the symmetrical bilateral projection of the optic foramen on a

Fig. 11 (skull 1,285).—In *A*, an optic foramen axis projector is seen in the left optic foramen. The movable central rod corresponds with the long axis of the foramen and indicates the pathway of the central pencil of roentgen rays in a properly taken oblique roentgenogram of this optic foramen. The dot on the external surface of the zygomatic bone marks the point of exit from the orbit of the foraminal axis and is a guide to the location of the foramen in a roentgenogram. In *B*, the large black dots on the calvarium indicate the points of exit of the posteriorly projected optic foraminal axes and also the points of entry of the central rogentgen rays in roentgenography of this skull in a postero-anterior oblique position.

single film without moving the head. His V-shaped device has the twofold function of fixing the head and holding a partly shielded casette close to the orbit and at right angles to the central rays of a laterally tilted roentgen tube. This device overcomes the objectionable obliquity of the film to the central rays when the roentgen tube is

11a. Goalwin, H. A.: Der Canalis opticus bei normalen und deformierten Schädeln, Ztschr. f. Augenh. **54**:191, 1924; footnote 5.

12. Camp, J. D., and Gianturco, C.: A Simplified Technique for Roentgenographic Examination of the Optic Canals, Am. J. Roentgenol. **29**:547 (April) 1933.

13. Dr. Goalwin and Dr. Pfeiffer.

tilted laterally and the casette is horizontal. This was a weakness of the technic of Palmieri.[14] Giordano[15] recognized this weakness and endeavored to correct it by his *craniografo* apparatus.

All the exposures were made with the same roentgen tube at a 27 inch (68.58 cm.) anode-film distance and a distance from 2½ to 3 inches (6.3 to 7.5 cm.) between the object (posterior opening of the optic foramen) and the film.

A craniophore was used which permitted fixing and holding a skull in any required position. To procure a roentgenogram taken directly along the axis of a foramen two devices were used: 1. A metal contrivance (figs. 11, 12 and 13) indicated where the foramen should appear in relation to the orbital entrance. 2. A centered, long wooden stylus, tapered at one end, fitted in the canal from the cranial entrance and indicated the pathway which the central roentgen rays should pursue (fig. 14).

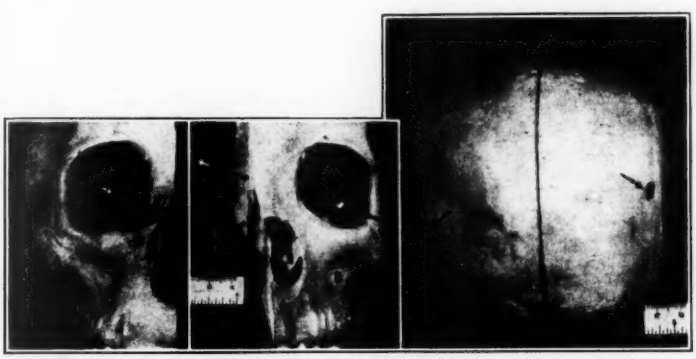

Fig. 12 (skull 2,242).—Views showing asymmetry of the skull and the location of the optic foramina: *A*, the right orbit, with an optic foramen axis projector in situ; *B*, location of the axis of the left optic foramen, indicated by an axis projector; *C*, points of contact of the posteriorly projected foraminal axes, indicated by the large black dots.

When the Goalwin method was used the various base lines and angles were drawn on the skull. Camp's localizer and Pfeiffer's device were obtained and utilized in their technic.

An assay of the results revealed that the method of Goalwin projected the foramen low down in the lower outer quadrant or into or below the lower margin of the orbit. It therefore produced considerable distortion in the outlines of the majority of foramina, which, as figure 9 shows, should be projected much higher. With Camp's device

14. Palmieri, G. G.: Tecnica per la proiezione simmetrica dei canali ottici dei due lati, in due tempi sudiuna stessa film, Riassunto communicaz. IX cong. ital. di radiol. med., 1930.

15. Giordano, Giovanni: Craniografo. Nuovo e simplice dispositivo per la radiografia simmetrica dei canali ottici e di altre parti della base cranica, Riv. di radiol. e fis. med. **3**:341 (May) 1931.

the foramen was seen in the lower outer quadrant but frequently appeared distorted owing to lateral as well as vertical displacement. With Pfeiffer's technic it was easy to show the foramen bilaterally in the lower outer orbital quadrant. However, the outlines were not sufficiently accurate to permit exact deductions, such as estimation of dimensions.

The three methods definitely projected the average optic foramen lower in the orbit than it appeared in the control, in which the central rays coincided exactly with the axis of the foramen. A lateral deviation, although less than the vertical, was also noted. Most roentgenograms showed evidence of both types of deviation, and some showed evidence of torsion. This situation excited curiosity concerning the possibility of estimating the actual diameters of an optic foramen from measurements of its outline on a roentgen film.

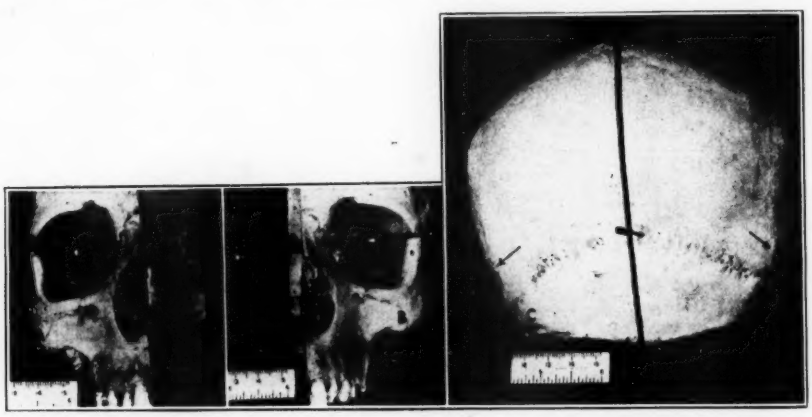

Fig. 13 (skull 1,162).—An example of nearly symmetrical optic foramina. The optic foramen axis projector and the black dots on the calvarium indicate foramina the axes of which are high in the orbit and low on the skull.

ESTIMATION OF THE SIZE OF THE OPTIC FORAMEN FROM A ROENTGENOGRAM

Goalwin [5e] stated:

> Measurement of the size of the optic canal in the living is done by means of roentgenography. . . . the true shape of the cross section of the optic canal is obtained, but not the true size. This is enlarged.

Pfeiffer [8] expressed the belief that the shadow of the foramen on a roentgenogram is an enlargement.

> In roentgenography when a small focus tube and 25 inch target film distance are used, the diameter of the shadow of the canal on the film is approximately 1 mm. larger.

Fig. 14 (skull 1,806).—Tapered, centered wooden stylus is seen in an optic foramen. This was used to indicate the pathway of the central roentgen rays when the skull was in the oblique position.

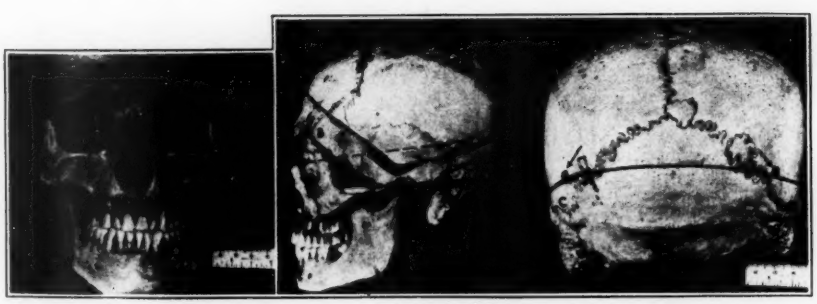

Fig. 15 (skull 424).—A brachycephalic skull. The axes of the optic foramina are above the midline of the orbit. The right is slightly closer to the lateral orbital margin. *A* shows an optic foramen axis projector in the right foramen. *B* shows a foramen projector in the left foramen; the broken black line indicates the foraminal axis in the horizontal plane. In *C*, the large black dots mark the points of incidence of the foraminal axes on the calvarium.

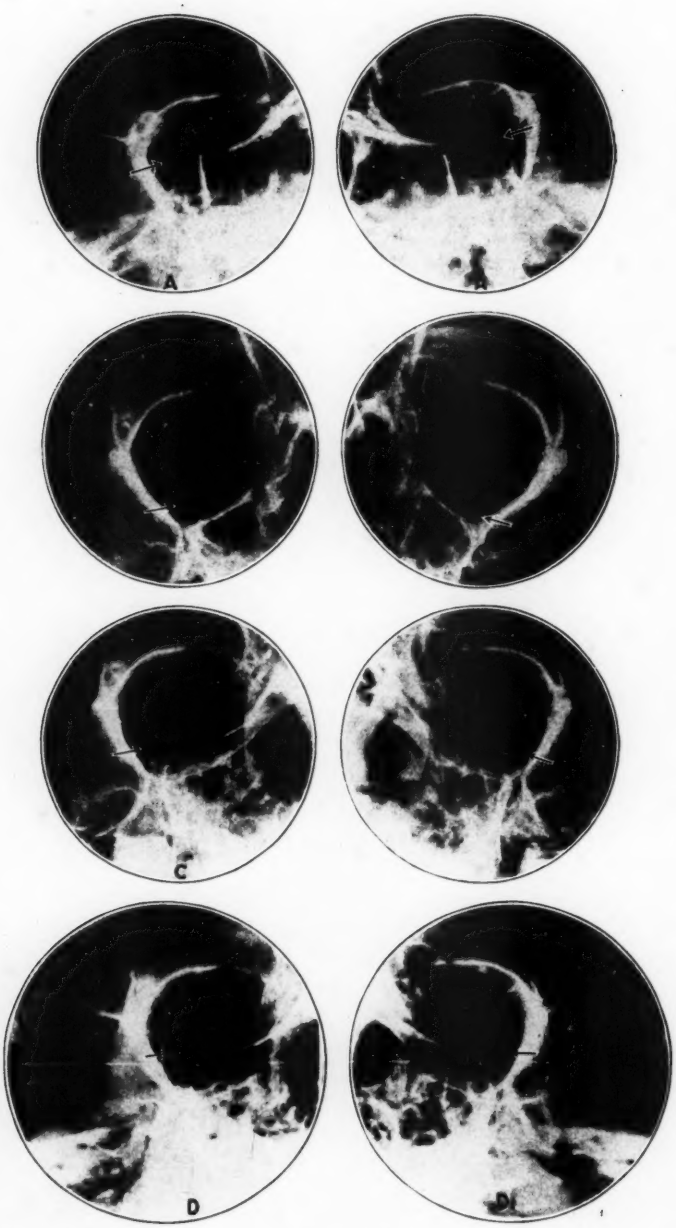

Fig. 16 (skull 424).—Roentgenograms in the oblique position. The optic foramen is indicated by the arrow. *A* is a normal control view, in which the central pencil of roentgen rays was alined with the long axis of each optic foramen. The other roentgenograms were taken by the following methods: *B*, by the method of Goalwin; C, by the method of Camp, and *D*, by the method of Pfeiffer.

Van der Hoeve [16] also calculated the diameter of the optic foramen from his roentgenograms, while White,[17] opined that "The diameter of an optic canal of 5 mm. would be enlarged about ½ mm. in the radiograph of a living subject."

There is a widespread impression that the minimum cross-section and the minimum diameter of the optic foramen are those of the posterior orifice. Goalwin [17a] stated: "Its minimum cross section is at the posterior end, where, therefore, the measurements should be made." [5e] Del Duca,[18] Senà,[19] Speciale-Piccichè [20] and others have expressed the same opinion.

To illustrate this point various types of foramina were chosen. The maximum and minimum diameters were measured at the posterior and anterior orifices and in the middle when the floor was sufficiently long to permit. An average of these diameters was obtained. In table 5 these measurements are compared with those derived from roentgenograms made along the known axis of the foramen (normal control) and by the methods of Goalwin, Camp and Pfeiffer.

A perusal of table 5 reveals several interesting facts, which are briefly summarized as follows:

1. The location of the smallest cross-section of the optic foramen was not constant. It was not always at the posterior orifice; neither was the shortest diameter.

2. The average of the maximum and minimum diameters was deceptive.

3. Variations in the caliber of the foramen were so frequent that its diameters at any given point did not present a complete picture. It was necessary to know at least the maximum and minimum measurements of several cross-sections to visualize the size of the foramen.

4. It was even more difficult to gage with a caliper the shape of the foramen. The planes of the maximum and minimum diameters were inconstant.

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16. Van der Hoeve, J.: Radiographie du trou optique, Bull. et mém. Soc. franç. opht. **38**:693, 1925.

17. White,[7] p. 127.

17a. Goalwin, H. A.: Ztschr. f. Augenh. **54**:191, 1924; One Thousand Optic Canals: Clinical, Anatomic and Roentgenologic Study, J. A. M. A. **89**:1745 (Nov. 18) 1927.

18. Del Duca, quoted by Giordano,[9] p. 805.

19. Senà, José A.: El conducto óptico, Semana méd. **1**:702 (March 3) 1932.

20. Speciale-Piccichè, P.: Il cannale ottico dal punto di vista radiologica, Ann. di ottal. e clin. ocul. **55**:769 (Oct.) 1927.

5. The roentgenographic outline was not always larger than the average or even larger than other diameters of the foramen.

NEW APPARATUS

It was necessary to devise certain apparatus [21] which consisted of:

1. A craniophore. This grasps the skull through the foramen magnum, or by half of the calvarium, allowing it to be held firmly in any required position (fig. 22).

TABLE 5.—*Comparison of the Maximum and Minimum Diameters of Optic Foramina in the Skull and Their Silhouettes on Roentgenograms* *

		Diameters in Skull				Diameters on Roentgenograms			
Skull Number		Posterior	Anterior	Middle	Average Maximum and Minimum	Normal Control	Goalwin Method	Camp Method	Pfeiffer Method
424	R	6.5×5.2	5.3×6.1	5.3×5.7	6.1 ×5.26	5.7×6.2	5.7×5.1	5.7×7.5	5.7×7.6
	L	5.6×4.8	5.4×5.9	5.6×5.2	5.7 ×5.1	5.8×6.2	5.7×8.0	5.5×7.3	5.6×7.3
1,340	R	6.7×4.3	4.7×5.5	4.6×5.1	5.76×4.5	4.9×5.5	4.8×6.4	4.8×5.6	4.5×5.8
	L	6.6×4.2	4.7×5.6	4.6×4.9	5.7 ×4.5	4.8×5.6	4.6×6.4	4.8×5.6	4.9×5.9
1,788†	R	6.1×4.2	4.5×6.2	4.2×4.2	5.5 ×4.3	4.3×4.2	†	3.9×5.1	4.6×4.3
	L	5.4×4.0	4.2×5.5	4.1×4.5	5.1 ×4.1	4.2×4.1	†	3.6×5.0	4.6×4.1
2,009†	R	6.0×4.1	3.8×5.0	4.4×4.1	5.1 ×4.0	3.9×4.2	4.7×5.8	3.9×4.7	4.5×4.7
	L	5.7×4.6	4.0×5.5	5.1×4.7	5.4 ×4.4	4.1×4.6	†	4.7×5.0	4.1×4.8
1,363	R	5.2×4.4	4.8×5.4	4.5×4.5	5.0 ×4.56	4.7×4.9	4.8×4.9	4.8×4.0	5.3×4.2
	L	5.0×4.6	4.4×5.0	4.4×4.6	4.86×4.46	4.5×4.7	4.6×4.7	4.1×4.0	4.6×4.5
1,205‡	R	4.4×5.4	4.5×6.2	4.5×5.8	5.8 ×4.46	4.5×5.6	4.1×5.6	4.1×5.9	4.6×5.5
	L	4.6×3.8	4.6×6.1	‡	5.35×4.2	4.7×6.6	4.2×5.4	4.0×6.5	4.8×6.4
1,651	R	5.6×4.4	4.6×5.2	4.7×4.5	5.16×4.5	4.4×4.8	4.0×5.7	3.5×4.4	4.4×4.9
	L	5.4×5.0	4.8×5.3	4.5×4.7	5.1 ×4.76	4.3×5.5	3.9×7.2	4.4×5.5	4.9×5.9
1,469	R	5.2×4.3	4.0×5.1	3.7×4.1	4.8 ×4.0	3.6×3.9	3.6×4.3	3.0×4.0	4.0×3.9
	L	4.7×4.1	4.0×4.6	3.8×4.2	4.5 ×3.96	3.6×4.4	4.2×4.5	4.0×2.6	3.6×4.1
1,185	R	6.0×6.4	5.6×6.8	5.3×6.4	6.5 ×5.6	4.7×6.6	4.8×6.8	4.4×6.2	4.8×7.2
	L	5.9×5.9	5.7×7.0	5.5×6.0	6.3 ×5.7	5.0×6.3	4.8×7.3	4.5×6.0	4.9×7.9
440	R	6.2×4.8	5.1×5.6	4.3×4.8	5.5 ×4.7	4.6×5.0	5.5×5.7	5.0×5.2	5.0×5.1
	L	5.8×4.4	4.9×5.8	5.2×4.4	5.6 ×4.56	4.7×4.6	4.4×6.2	4.7×5.3	5.0×5.3

* The measurements are expressed in millimeters. The first diameter is approximately that of the horizontal plane, the second, that of the vertical; hence the maximum reading does not always appear first in the table, except in the column headed "Average Maximum and Minimum."

† The outline in skull 1,788 and on the left side of skull 2,009 was too obscure for measurement by the Goalwin method.

‡ No measurement was made of the middle diameters on the left side owing to the short floor.

2. A caliper. This makes it possible to measure internal and external diameters up to 10 mm. It has a 0.1 mm. scale on a dial. The tip of the blades can enter apertures as small as 2 mm. This instrument

21. This apparatus was made by Mr. Joseph Cherney in the anatomic workshop of the laboratory of anatomy at Western Reserve University.

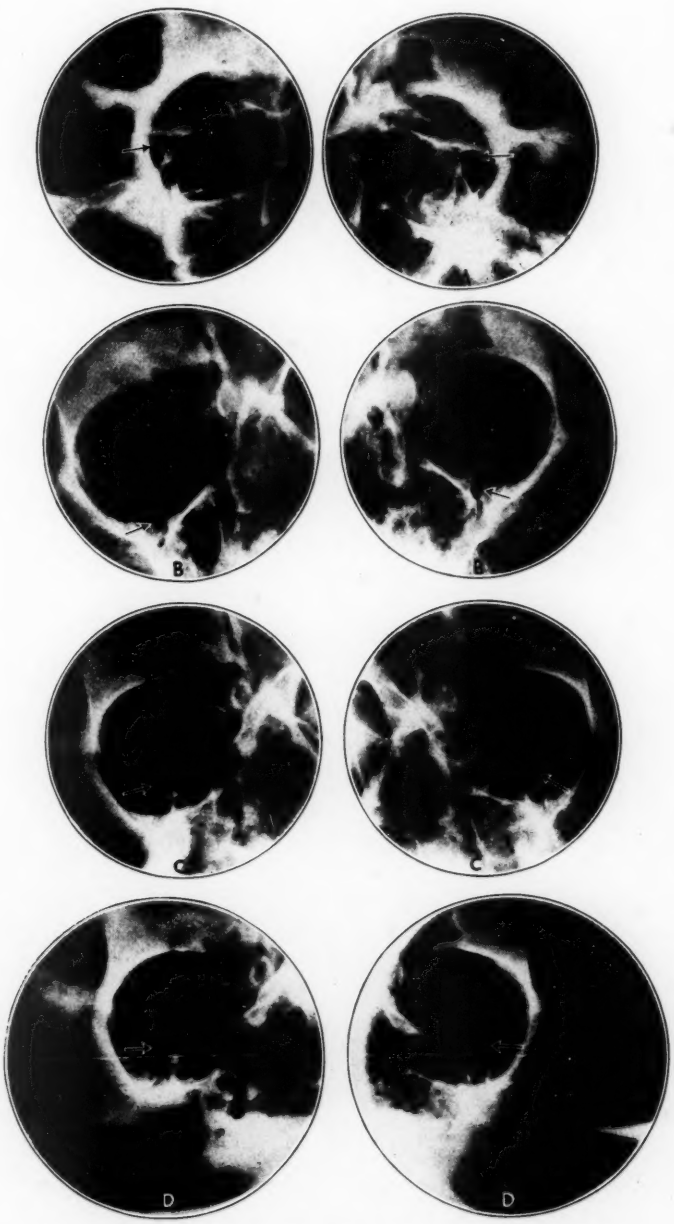

Fig. 17 (skull 1,185).—A dolichocephalic skull with large optic foramina and asymmetrical axes, the right lower and near the outer orbital wall. The optic foramina as seen on the roentgenograms are indicated by the arrows. *A* is a normal control view. The other roentgenograms were taken by the following methods: *B*, by the method of Goalwin; *C*, by the method of Camp, and *D*, by the method of Pfeiffer.

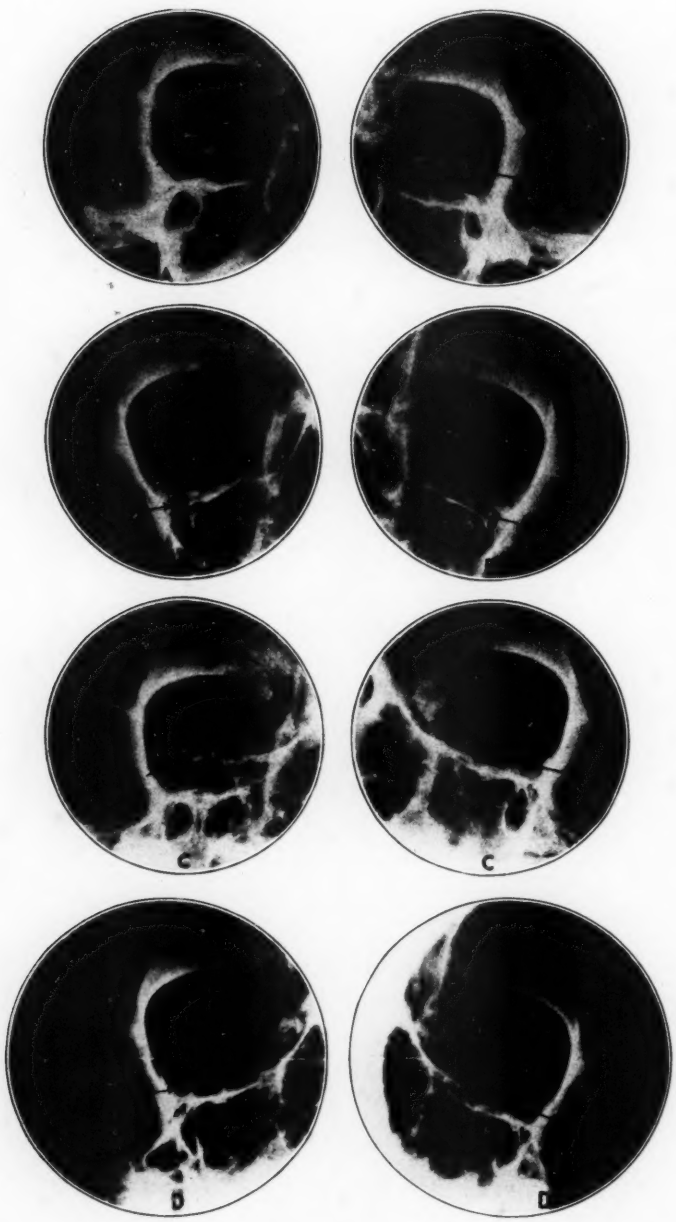

Fig. 18 (skull 1,340).—A dolichocephalic skull. The optic foramina project into the lower outer quadrant of the orbit near the inferolateral margin. This is the region where, according to some authors, most foramina should appear. The optic foramina as seen on the roentgenograms are indicated by arrows. *A*, is a normal control view. The other roentgenograms were taken by the following methods: *B*, by the method of Goalwin; *C*, by the method of Camp, and *D*, by the method of Pfeiffer.

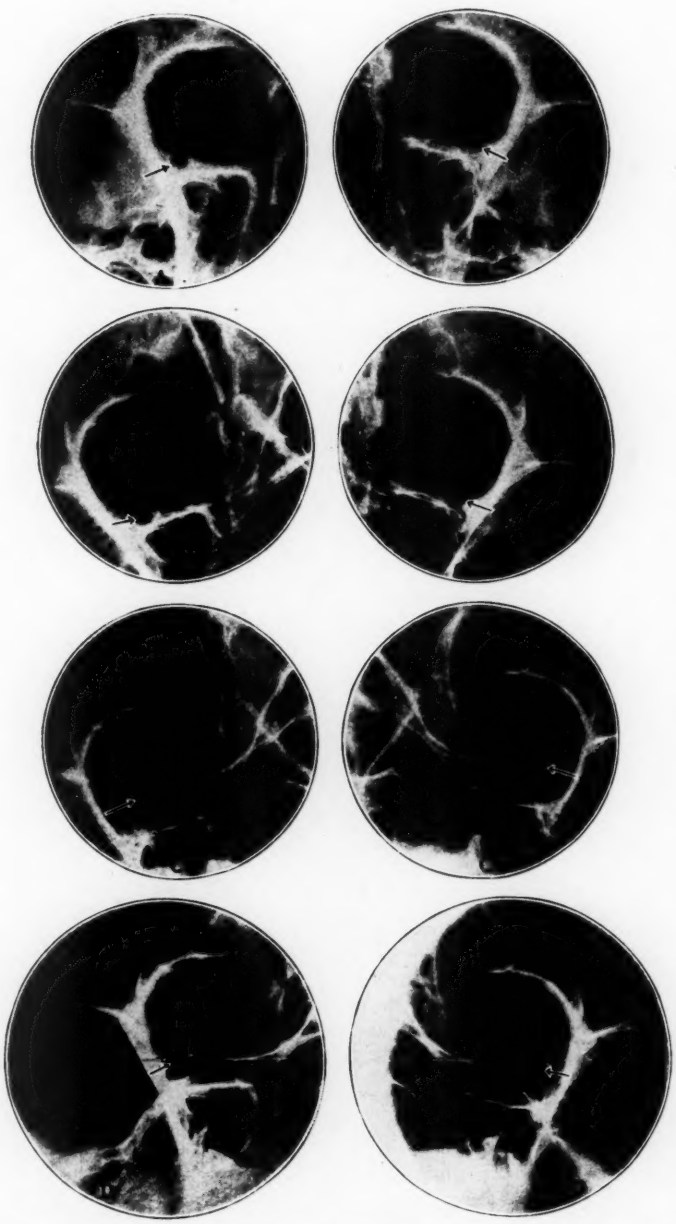

Fig. 19 (skull 1,363).—A dolichocephalic skull. The right optic foramen projects into the zygomatic bone; the left is tangent to the lower orbital margin. The optic foramina as seen on the roentgenograms are indicated by the arrows. *A*, normal control. The other roentgenograms were taken by the following methods: *B*, by the method of Goalwin; *C*, by the method of Camp, and *D*, by the method of Pfeiffer.

was devised to measure the diameters of foramina and narrow tongues of bone (fig. 23).

3. An optic foramen axis projector. This consists of a 6 cm. frustum of a cone tapered from a 2 mm. diameter at the tip to 6 mm. at the base. It is drilled in the center to allow the passage of a close-fitting $\frac{1}{16}$ inch (0.16 cm.) drill steel rod (fig 11 *A*).

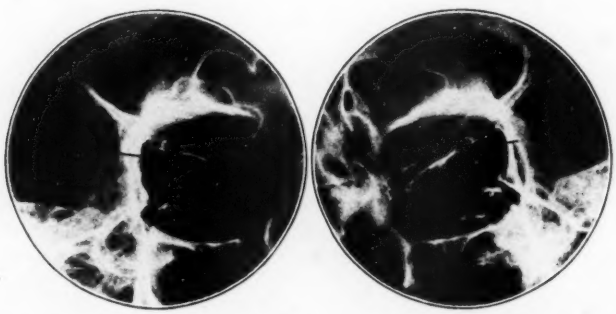

Fig. 20 (skull 1,788).—A mesocephalic skull. All three clinoid processes are united bilaterally by osseous bridges. The roentgenograms of the optic foramina show the absence of interference from the clinoid bridges. The optic foramina, indicated by arrows, project high into the outer orbital quadrant.

Fig. 21 (skull 2,207).—Roentgenogram of the left optic foramen, showing a rare condition, namely the absence of the floor of the foramen.

4. Orbital quadrant. This is composed of transparent stiff celluloid and is of a size to cover the largest orbit. It is divided into quadrants and subdivided into 5 mm. squares. The horizontal and vertical midlines are used as guide lines. Enough of one quadrant is removed to allow for the nasal bone. The device is reversible and can be used over either orbit (fig. 10).

ADDITIONAL TECHNIC [22]

To tilt the axis of an optic foramen a given number of degrees a long, tapered stylus was inserted in the lumen posteriorly and the angulation measured by the stationary goniometer of Martin.[23]

To make the results of the projection of the axis of the optic foramen uniform and reproducible the standard anthropometric points and measurements of the orbit described by Martin [23] were adopted. Those used were:

1. The maxillofrontal point (Martin [24]), the point where the fronto-maxillary suture intersects the crista lacrimalis anterior or that crest prolonged.

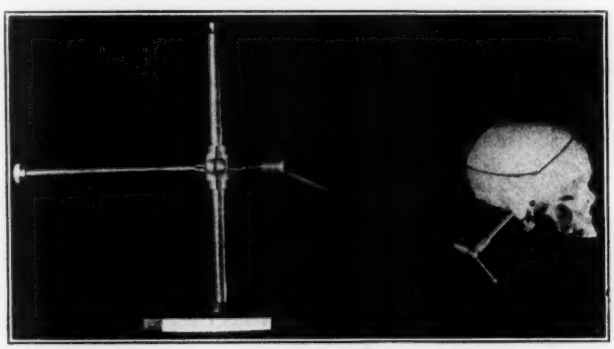

Fig. 22.—A craniophore. This instrument holds a skull or half a skull in any required position.

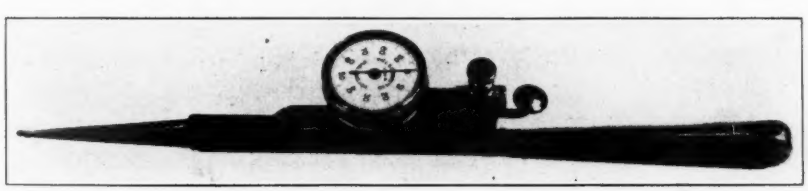

Fig. 23.—A caliper. This instrument measures internal and external diameters up to 10 mm.

2. The ectoconchion (Martin [25]), the point on the lateral margin of the orbit determined by the long axis of the orbit which runs from the maxillofrontal point parallel with the upper orbital margin.

22. To secure accuracy and validate the data, W. H. Sassaman, M. A., checked all the observations. All the roentgenograms were made by W. Kuenzel, M. A., roentgenologist to the laboratory of anatomy and associated foundations, Western Reserve University.

23. Martin, R.: Lehrbuch der Anthropologie, ed. 2, Jena, Gustav Fischer, 1928, vol. 2.

24. Martin,[23] p. 620.

25. Martin,[23] p. 621.

3. The orbital width (Martin[26]), which is measured from the maxillofrontal point to the ectoconchion in such a way as to take this long diameter as nearly as possible parallel with the upper orbital margin and to divide the orbital opening into halves.

The procedure was to apply the celluloid scale with its horizontal midline extending from the maxillofrontal point parallel with the upper margin of the orbit through the ectoconchion. The scale was adjusted until the vertical midline divided the orbit into an outer and an inner half and the vertical and horizontal midlines separated it into four quadrants (fig. 10).

The central rod of the foramen axis projector, in situ, was extended anteriorly until it touched the celluloid quadrant. The point of contact was noted and recorded.

The constants of the roentgen work were: the same roentgen tube, an anode-film distance of 27 inches, an object-film distance of from 2½ to 3 inches, a cone 10 inches by 3 inches (25.4 by 7.6 cm.), a current of 10 milliamperes and 71 kilovolts, an exposure of five seconds for open skulls, and from eight to nine seconds for closed skulls, no screens and a paper film holder.

SUMMARY

A study was made of the optic foramen in 2,187 human skulls of known origin. Two thousand and sixty-four were dried and 123 wet specimens; 1,354 were from white persons, and 833 from Negroes; 1,745 were from males and 442 from females.

Aberrations of the clinoid processes of the sphenoid bone were noted because of their proximity to the optic foramen. The first, the clinocarotid or anterior interclinoid canal (traversed by the internal carotid artery), formed by a bony union of the anterior and middle clinoid processes, was present in 34.84 per cent of the craniums. The second, an osseous bridge attached to the posteriod clinoid process, was found in 8.68 per cent of the specimens. In 0.86 per cent it united the posterior and anterior clinoid processes only and formed the common interclinoid canal of Gruber. In 7.82 per cent all three clinoid processes were bridged, thereby forming a posterior interclinoid venous canal.

The incidence was recorded of a foramen or sulcus in the floor of the optic foramen capable of housing the ophthalmic artery. A foramen of this kind was noted in 0.22 per cent of the skulls and a sulcus in 1.64 per cent.

The effect of the foregoing aberrations on roentgen delineation of the optic foramen was investigated. The interclinoid osseous bridges did not encroach on the lumen of the foramen, and ophthalmic foramina

26. Martin,[23] p. 659, measurement 51.

or sulci could be seen clearly in undistorted, properly taken roentgenograms.

The relationship between the projected long axis of the optic foramen and the plane of the orbital rim or entrance (aditus orbitae) was studied in 1,000 foramina, in 500 skulls. An axis did not appear in the mesial half of any orbit. The greatest incidence was along the horizontal midline at the junction of the upper and lower outer orbital quadrants. The influence of race and sex was noted.

A method was devised to obtain exact roentgenograms of the optic foramen in a dried skull. Observations were made of the results obtained when the skull was tilted a known number of degrees. Roentgenograms of the optic foramina were also made by the methods of Goalwin, Camp and Gianturco and Pfeiffer.

The maximum and minimum diameters of various types of optic foramina were measured at the posterior and anterior orifices and in the middle. An average of these diameters was obtained. These measurements were compared with those obtained from roentgenograms made along the known axis of the foramen (normal control) and by the methods of Goalwin, Camp and Pfeiffer.

COMMENT

This study shows that: 1. The outline of the average optic foramen, on a postero-anterior oblique roentgenogram should be projected higher in the orbit than has been the practice with many observers. Probably none will appear in the mesial half of the orbit. Seventy-nine per cent of projected foramina are found within the orbital aperture and within the outer (lateral) half of the orbit. The remainder (21 per cent) are tangent to or behind the outer or lower orbital margin.

2. Roentgenograms of optic foramina showing an irregular outline and inclusion of shadows other than those of the posterior roof should be retaken.

3. The roentgen outline is not always larger than the average diameter, or even larger than other diameters, of the optic foramen.

4. It is not possible as a routine to calculate the size and shape of the optic foramen accurately from its shadow on a roentgenogram.

5. Variations in the caliber of the optic foramen are so frequent that its diameters at any given point do not present a complete picture. It is necessary to know at least the maximum and minimum measurements of several cross-sections to visualize the size of the foramen.

6. The location of the smallest cross-section of the optic foramen is not constant. It is not always at the posterior orifice; neither is the shortest diameter.

In view of the foregoing observations it is believed that interpretations of roentgenograms of the optic foramen should be expressed as possibilities and probabilities and not as certainties.

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ABSTRACT OF DISCUSSION

Dr. N. W. Ingalls, Cleveland: Late in the second month of embryonic life, the central basilar portion of the chondrocranium makes its appearance underneath the brain. Forward extensions of this cartilaginous primordium of the skull form the nasal capsule and septum and the inner wall of the orbit; lateral outgrowths give rise to the orbitosphenoids and alisphenoids in front and to parts of the temporal and occipital bones behind. During this growth and extension, the new-formed, advancing cartilage adapts itself to many structures already present, more particularly the vessels and nerves on the ventral surface of the brain. As these earlier structures become surrounded, or even enveloped, by the growing cartilage, there arise corresponding foramina or canals, at first cartilaginous, later bony. Just as the structures traversing these openings are primary and also somewhat variable, so the later, adult structures are not always the same or even symmetrical on the two sides. The extent to which both cartilage and bone adapt themselves is well shown in the bony labyrinth and facial canals.

Among the structures thus enveloped is the optic nerve which is surrounded by the base of the orbitosphenoid. The optic foramen thus formed is, like many other foramina, relatively large, and only later, when definite relations are more nearly attained, does one find here, as elsewhere, a more exact conformity of the bone to the enclosed structure.

In these early stages one begins to encounter that individual variation so conspicuous later, and even the two sides of the skull may present minor differences. The most significant observation, however, is the extent to which the supporting skeletal tissue, whether bone or cartilage, adapts itself to the particular requirements; but rarely, if ever, except in pathologic conditions, does either tissue encroach or press on the structures transmitted. Not only do the numerous foramina vary, but the configuration and the extent of cartilage are subject to individual variation throughout development, and this variability is especially conspicuous in the region of the basisphenoid and orbitosphenoids, the clinoid process and the sella turcica. At these sites may be found in the cartilage the variations or anomalies which later appear in the bone, with occasional contributions from bone formation in the dura.

None of the numerous variations around the sella turcica and the optic foramen, anomalous bony processes or foramina, need be in any sense pathologic, nor is there any reason to believe that they are modified materially by age. They are rather examples of the inherent variability of all parts of the body. Although comparatively little is known about variability in embryonic life, all of the evidence indicates that variability is present in the embryo as in the adult, and if it is apparently less, this is only because the opportunities are not so great.

While the bases of the brain and skull are relatively constant in their morphologic characterstics and mutual relations, the newer, cerebral portions of the brain and the face and vault of the skull are subject to great individual, and often racial, differences. This applies to the orbit and to the relations of the optic canal to the orbit. The natural variations in the size, form, length and axis of the canal brought out so clearly by Dr. Keyes are further complicated by the wide range of differences in the orbit itself, and one need not be surprised if the determination of the axis of the canal in the living subject is not so simple as one could wish.

The problem at present is the orientation of a comparatively short, simple but variable canal, set in variable surroundings, to the outside of the head and face, more specifically to the orbital opening. At first the orbit has little more than an incomplete inner wall and a bit of roof behind; only later are the floor and the outer wall formed. The orbit is relatively large and high and appears more lateral in the fetus and early in life; later it becomes lower, especially in the male, and the lateral wall becomes more extensive. The orbits, like the skull above, are large in the infant and young child, and although they do not increase in size like the rest of the face, they are intimately involved in facial growth and are subject to great individual differences in size and shape and in their relations to each other, to the base of the skull and to the optic foramina.

Like the two halves of the skull, the two orbits are rarely symmetrical. The orbital aperture varies in its absolute dimensions as well as in its shape and in the relation of height to width, tending to be low in broad faces and higher in long faces; the transverse axis may be more or less inclined and its outer end some distance behind the inner; likewise, the depth of the cavity and the length of the walls vary, and the median wall may be longer than the lateral; always close to and often in line with the median wall, the optic foramen may lie internal to or some little distance lateral to this plane as it may also be higher or lower as seen through the orbital opening.

All these natural or normal variations are, in a way, aggravated by the small size of the orbit and particularly of the optic canal and its immediate surroundings. Small absolute differences in dimensions, relations, angles, etc., may easily assume significant if not serious proportions, particularly under the conditions imposed by roentgenologic examination.

Functionally, the variations in the optic canal appear to be of little significance, provided there is not an attendant abnormal condition or neighboring area of pathologic change. One may compare the optic with the much smaller and longer facial canal; both are potentially sources of trouble, but both are large enough to be adequate for their purpose unless abnormal, secondary conditions arise; then the thin arachnoid space around the optic nerve is probably of little service in preventing or mitigating any harmful rise in pressure.

DR. RAYMOND L. PFEIFFER, New York: Dr. Keyes' report represents a critical and laborious study of variations which are not generally appreciated and which are of great importance to those interested in the roentgenology of the skull.

The question is frequently asked: "Of what importance are films of the optic canal?" At the Eye Institute of New York stereoscopic films of the canals were made as a routine by my method for all patients coming to the x-ray department. In a series of over 600 skulls 8 per cent showed interesting anatomic variations and pathologic changes.

The principal indications for roentgenography of the optic canals are exophthalmos, papilledema, optic atrophy, obscure changes in the visual field, vascular disease and traumatic disease of the orbit. Because in my experience disease of the paranasal sinuses has proved to be an infrequent cause of ocular disease, it is not listed as an indication. The relationship of the sphenoid sinuses to the canals is, however, interesting indeed. The canals have been found to pass through these sinuses and be entirely surrounded by air space.

The precise measurement of a canal is not important, for the canals vary greatly in size in normal skulls, and the sizes of the canals on the roentgenograms depend on many factors in the technic of production of the films. Of great importance is the comparison of the two canals, which requires that they be projected as symmetrically as possible. Slight variations in the axes of the canals may mislead or confuse, and therefore the canals should be projected in stereoscopic views. The method and device used should securely immobilize the head, project the canals symmetrically, reproduce accurately previously projected films, work satisfactorily for routine use and, by means of a simple adjustment, change the alinement of the canals when needed.

LECTURES ON MOTOR ANOMALIES OF THE EYES

III. PARALYSES OF THE CONJUGATE MOVEMENTS OF THE EYES

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ASSOCIATED PARALYSES OF THE PARALLEL MOVEMENTS

The disorders of the movements of the eyes caused by lesions of peripheral origin, i. e., of the nuclei and of the individual motor nerves, have been discussed previously.[1] If a lesion is situated above the nuclei, injuring the supranuclear apparatus, i. e., the pathways between the nuclei and the cortical, the subcortical or the vestibular centers, paralysis of the associated muscle groups of both eyes occurs as a rule. The few exceptions to this rule are the paralyses caused by a lesion of the posterior longitudinal bundle or in the immediate neighborhood of the nuclei of the oculomotor nerves. Such a lesion can deprive one internal rectus muscle of the faculty of adduction in lateral movements, leaving intact its convergence function, or it can make both elevator muscles of one eye unable to produce a voluntary elevation without disturbing the involuntary elevation noted in Bell's phenomenon. Theoretically, supranuclear paralysis of the other individual eye muscles are quite conceivable, but there is no way of differentiating between them, because the other muscles do not act as parts of different mechanisms in anything like the same degree as the internal rectus muscle acts in lateral and in convergence movements or as the elevator muscles act in obeying the elevation impulse and the impulse to shut the eyes. Therefore, but for the few exceptions just mentioned, the diagnosis of a supranuclear lesion is chiefly based on bilateral and equal paralysis of associated muscle groups.

I shall discuss first associated paralysis of the parallel lateral movements, the symptoms of which have been studied mostly in paralyses caused by lesions of the association pathways either within or near the pons and the region of the fourth ventricle. In an uncomplicated case of this type, both eyes, being unable to move to the right, for instance, are slightly deviated to the left. This deviation induces the patient to turn his head a little to the right in order to look at an object straight in front of him. No diplopia is present because of the equality of the

1. Bielschowsky, Alfred: Lectures on Motor Anomalies of the Eyes: I. Physiologic Introduction, Arch. Ophth. **12**:805 (Dec.) 1934; II. Paralysis of Individual Eye Muscles, ibid. **13**:33 (Jan.) 1935.

deviation on the two sides. When the patient is ordered to look at an object on the right, neither eye can move beyond the midline, or if the movement is not lost but only weakened, the restriction of the movement is equal on the two sides, so that binocular single vision is not disturbed. In such a case jerky nystagmus to the right reveals the repeated futile efforts to direct the eyes toward the peripherally located object. Strict proof of the supranuclear origin of such a paralysis is given by the intact convergence function of the left internal rectus muscle, which is unable to turn the left eye in only when a parallel movement to the right is called for. From this behavior it must be concluded that the nerve to the left internal rectus and its nucleus, as well as the pathway which descends to the latter from the convergence center, are intact, but that the pathway descending from the cortical center governing the associated movement to the right has been interrupted. However, it is not yet known whether the paralysis of the right external rectus muscle is also caused by a supranuclear lesion. It is quite possible that a lesion of the right abducens nucleus involves the posterior longitudinal bundle as well, so that the left internal rectus loses its associated function with the right external rectus. This can be decided when the response of the right abducens nerve to a vestibular stimulus is examined.

In 1901 I [2] observed a patient with an acute encephalitis centralized in the pons which had produced an associated paralysis of the parallel movement to the left, the convergence function of both of the internal rectus muscles being undisturbed. The patient was unable to move her eyes to the left at a word of command and was also unable to look at an object situated in the left half of her visual field. In the face of such an apparently complete inability of the eyes to move to the left, the result of further examination was all the more surprising. While the patient was fixing an object straight in front of her I rotated her head with a sudden jerk to the right. The result was that both eyes moved to the left to a nearly normal extent, but immediately after the rotation of the head had been completed the eyes went slowly and involuntarily back to their original position—a proof of the reflex character of the ocular movement that had just been produced. In a second test, while the patient was fixing an object straight in front of her I rotated her head slowly to the right. Again the eyes moved to the left as far as in the first test, but this time they were able to maintain fixation and remained directed to the left as long as the head was kept rotated to the right, a proof that this time the ocular movement was caused not by a vestibular reflex action but by an impulse of cortical

2. Bielschowsky, A.: Das klinische Bild der assoziierten Blicklähmung und seine Bedeutung für die topische Diagnostik, München med. Wchnschr. **50**:1666, 1903.

origin. The same effect was obtained when, instead of rotation of the patient's head, the object looked at was carried slowly to the left. Both eyes maintained fixation and followed the object as far as in the other tests and remained directed to the left for a considerable time. The ocular movement to the left produced in such a manner I call *Führungsbewegung;* Duane calls it the "following movement." It arises from the same innervation of cortical origin as the ocular movement in the second test. I shall try to explain later how the different results arise which are obtained in making use of the described tests.

By far the most frequent of the associated paralyses of the vertical movements are the paralyses of the elevator muscles, next frequent are the paralyses of the elevators and depressors, and the rarest are the paralyses of the depressors. In 1906 Steinert and I [3] reported on a series of cases of paralysis of the associated vertical movements, the characteristic signs and symptoms of which are shown in the following case report:

A man aged 48 had paralysis of the elevator and the depressor muscles following a paralytic stroke. He was unable to look either up or down when ordered to and was likewise unable to fix an object in his visual field above or below the horizontal plane. When he sat opposite a vision-testing card so that the largest letter was on a level with his line of vision, he could read the largest letter but was unable to read the letters in the next line below. That this was not due to a deficiency of vision was easily proved. If the card was gradually raised the patient could read each of the lower lines as soon as it came up to the level of his line of vision, thus showing that he had a normal visual acuity. But after he had read the lowest line he was unable to read the larger letters, because they were at that time situated above the horizontal plane.

The same result was obtained if, instead of raising the test card, prisms of increasing strength were placed base down before the patient's eyes. He was then able to read the lower lines. When the prisms were placed base up they helped him to read the letters above the horizontal plane. But when a card with the largest letters on top was brought to the same horizontal plane as the patient's line of vision, a prism of 10 degrees placed base up before each eye made the letters illegible; similarly, if the prisms were placed down before his eyes, the letters on the lowest line became illegible. In other words, the patient was unable to compensate a prismatic displacement of even 5 degrees by a corresponding vertical ocular movement. In striking contrast to this result was the ability of the patient's eyes to follow an object to an almost normal extent when it was slowly moved up and down and to remain in the elevated or depressed position for a considerable time. But from either position they were unable to return to the horizontal plane without being led by the guiding object.

Another interesting experiment was as follows: The patient sat opposite a vertical tangent scale, the fixed lamp being on a level with his eyes. He was unable at the word of command to direct the visual lines at one jump to one of the more distant numbers, for instance to the number 5 above or below the

3. Steinert, H., and Bielschowsky, A.: Physiologie und Pathologie der vertikalen Blickbewegungen, München. med. Wchnschr. **53**:1613, 1906.

lamp, whereas he succeeded in turning his eyes gradually and with a noticeable effort from the lamp to the number 1, from 1 to 2 and so on up to the upper or down to the lower end of the scale. It was not a normal movement but a slow creeping of the eyes along the successive numbers above or below the lamp. A quick vertical movement of the patient's eyes up or down to their normal limits could be produced only when his head was turned passively with a sudden jerk upward or downward. In Germany this behavior is called *Puppenkopfphänomen* (doll's head phenomenon). But as in paralysis of the lateral movements, immediately after the cessation of the passive rotation of the head, the eyes glided back automatically to the original middle position.

Finally, Bell's phenomenon was looked for. When the patient was asked to shut his eyes they went upward to the normal extent.

In some cases Bell's phenomenon is the only proof of the supranuclear origin of paralysis of the vertical movements and of the integrity of the nuclei. An example of this is shown in the accompanying photographs of a patient with associated paralysis of the elevator muscles caused by a tumor of the corpora quadrigemina. The usual position

A B C D

Associated paralysis of the elevator muscles caused by a tumor of the corpora quadrigemina. A, in the usual position of the eyes, both visual lines are equally depressed. *B*, the visual lines can be raised voluntarily or at a word of command only to the horizontal plane. *C*, they cannot pass it even if the fixed object is slowly moved upward. *D*, only in Bell's phenomenon, if the patient is asked to close his eyes, is a maximum elevation obtained, which is proof of the supranuclear localization of the lesion.

of the eyes is seen in the first photograph (*A*); both visual lines are equally depressed. Neither at the word of command (*B*) nor when the fixed object was slowly moved upward (*C*) could the visual lines pass the horizontal plane. The Puppenkopfphänomen was also absent, whereas Bell's phenomenon was perfectly demonstrable (*D*). After the tumor had been extirpated the first faculty to return was that of following an object which was slowly moved upward; the last was that of elevation at a word of command.

What is the significance of the various results obtained by examining the associated paralyses by means of the methods just described? They make it possible not only to decide that a paralysis has been caused by a supranuclear lesion but also, at least to a certain degree, to localize

the lesion more precisely, since it can be determined which of the various pathways descending to the nuclei are intact and which have been injured. If an associated group of muscles of both eyes, for instance the elevator muscles, is unable to perform a voluntary or commanded movement but reacts promptly when the patient's head is rotated passively by a sudden jerk downward, one must infer that the posterior longitudinal bundle which conveys the vestibular stimulus to the oculomotor nuclei is intact.

Some authors have doubted my interpretation of the vestibular origin of the ocular movements obtained by the passive jerky rotation of the head. Roth,[4] who published similar observations, attributed the reaction of the paralyzed muscles in response to the passive rotation of the head to a facilitation of the work to be done by the paralyzed muscles. If this explanation is correct, rotation of the head should enlarge the field of fixation in cases of paralysis of peripheral origin also, but this can never be observed. Moreover, I have seen patients with associated paralysis in whom the result of the head rotation test was at first positive but became negative when in a later stage the tumor had attacked the nuclei. According to other authors the reaction of the paralyzed muscles in this test is not caused by vestibular stimulation, as I believe it to be, but is a "following movement," since it is obtained also by a slow movement of the sighted object. They point to the fact that the fixation tendency plays an important part in the head rotation test. In response it must be said that in some cases the paralyzed muscles, responding only to a vestibular stimulus, react promptly when the patient's head is passively rotated by a sudden jerk, whereas the eyes are not able to follow the fixed object when it is slowly moved to the paralyzed side or when the head is slowly turned to the opposite side. In other cases in which both the following movement and the reflex movement of vestibular origin were present in the first stage of observation, the following movement was lost later; on the other hand, if the following movement was absent at first, it returned during the stage of improvement. That the fixation tendency does not play the principal part in producing this reaction of the paralyzed muscles when the head is turned by a sudden jerk is further proved by the positive results of this test obtained by Gordon Holmes in blind persons with associated paralyses. In general the fixation of a given object is necessary only to prevent voluntary movements during the test.

Bárány's[5] tests for ascertaining the vestibular excitation present an essential improvement in the examination of patients with associated

4. Roth: Demonstration von Kranken mit Ophthalmoplegie, Neurol. Centralbl. **20**:921, 1901.

5. Bárány, R.: Reflektorische vestibuläre und optische Augenbewegungen und ihre Bedeutung für topische Diagnostik der Augenmuskellähmungen, München. med. Wchnschr. **54**:1072, 1907.

paralyses. They can be used for the bedridden or the somnolent patient, and, further, since the vestibular stimulus continues for about a minute its effect can be observed much better than in the head rotation test, which produces only a momentary excitation. I cannot go into detail here concerning the various Bárány tests. The patient sits on a revolving stool and is rotated about ten times. The vestibular stimulation which results from the current thus set up in the endolymph of the semicircular canals produces a labyrinthine (jerky) nystagmus toward the side of rotation; a sudden arrest of the rotation brings about a secondary nystagmus in the opposite direction lasting for from thirty to forty seconds, provided the vestibular apparatus and its connections with the nuclei of the paralyzed muscles are intact. If the patient is bedridden, somnolent or a young child, the caloric test is preferable. After it has been ascertained by examination that the ear is normal, as it must be, water at 25 C. is syringed into the right auditory passage; from ten to twenty seconds later labyrinthine nystagmus toward the opposite side arises if the vestibular apparatus is intact. If the patient is unconscious there will be no nystagmus but only a slow movement of labyrinthine origin toward the same side; i. e., in the example chosen it will be toward the right, even if there is an associated paralysis of dextroversion of supranuclear origin. If, however, the right eye lags behind, it must be inferred that the nucleus of the abducens nerve or the nerve itself is injured.

As long as the paralyzed muscles respond to vestibular stimulation it may be assumed that there is integrity of the nerves, of their nuclei and of the pathways connecting them with the vestibular apparatus. Although in many instances a patient with this type of paralysis is unable to turn his eyes either at a word of command or spontaneously in the direction of an object or of a sound, he can do so if he is at first confronted with an object and then asked to follow it in the required direction. How this following movement is released and where the innervation concerned arises is still an unsolved problem. Since there are several centers in the cerebral cortex—in the frontal, in the occipital and in the temporal lobe—which are concerned with the associated movements, some authors have supposed that the innervations causing the following movement and the willed or commanded movement are derived from different centers and pathways, so that a lesion which interrupts the pathway descending from the frontal lobe makes the voluntary movement disappear while the following movement may not be disturbed. However, many patients, although they are able to follow a moving object, lack the ability to make an attraction movement, i. e., to turn the eyes toward an outlying object the image of which situated in the periphery of the retina attracts their attention. Both kinds of movements belong to the so-called psycho-optic reflexes because.

being produced by visual stimuli, they are performed more or less instinctively, so that one is justified in placing the origin of these movements in the occipital lobes. In spite of the fact that only the attraction movement can be lost and not the following movement, which remains more or less intact, it is not necessary, in my opinion, to assume the existence of separate centers and pathways for them. The patient whose case was cited as an example of associated paralysis of vertical movements was unable to overcome a prism of 10 degrees, base down or up, by elevation or by depression of the visual lines, nor was he able to turn his eyes from the fixed point to an object the image of which was situated 5 degrees above or below the retinal centers. But he brought about vertical movements of 10 degrees and more when he was given prisms of gradually increasing strength, beginning with 1 or 2 degrees, or when he was induced to turn his eyes to an object which was situated near the fixed point, either above or below it, and from that object to another nearby, so that the eyes performed a kind of climbing along a vertical series of objects that were close together. This behavior strongly suggests a comparison with fusion movements which are independent of the will. From testing the vertical duction power, for instance, it is known that the patient is not able in the beginning to overcome a prism of 5 degrees or more. But by beginning with a prism of 1 or 2 degrees and gradually increasing the strength of the prism he can be induced to overcome from 8 to 10 degrees by the appropriate vertical divergence. Such a fusion movement as well as the following movements in cases of associated paralysis may be explained in the same way. The nearer to the fovea the image which attracts attention is situated, the stronger is the motor excitation that is produced. If a weak prism is placed base out before one eye, adduction takes place involuntarily, whereas in spite of causing diplopia a strong prism in the same position does not produce that movement.

If the pathway descending from the occipital center is injured but not fully interrupted, the patient may be unable to perform an attraction movement of large extent, but he is able to make a small movement induced by the excitation of a paracentral retinal point. When the fixed object is moved slowly or the strength of the prism is increased little by little so that the retinal image of the fixed object moves from the fovea to a place in its proximate vicinity, a new small compensating movement results which finally becomes of considerable extent. This theory of mine is still to be proved, but it takes into consideration all of the clinical signs and symptoms, especially the fact that the following movement can be produced only by carrying the object slowly across the field of fixation or by turning the patient's head slowly in the opposite direction. Unlike the sharp jerking of the head, such slow rotation causes no vestibular reflex movement.

According to the particulars which can be ascertained by the various methods of investigation, it may be possible to make an approximate localization of the lesion causing the associated paralysis in an individual case. "Pseudo-ophthalmoplegia," so-called by Wernicke since he saw it as a part-manifestation of pseudobulbar paralysis, displays the following characteristics: The patient is unable to move his eyes at a word of command, but they are moved involuntarily in states of emotion or if the patient is interested in an object or in a sensory excitation deriving from an object. The following movements can be produced provided the patient's attention is attracted to the moving object. The reflex movements of vestibular origin are undisturbed. In some of these cases the whole oculomotor apparatus, including the cortical centers, is undamaged, the lesion being transcortical and damaging the connections between the frontal oculomotor centers and other parts of the cortex. Similar symptoms occur in diseases of the extrapyramidal system.

More frequently one encounters a second group of associated paralyses, characterized by the inability not only to move the eyes in a certain direction either voluntarily or at a word of command but also to move them toward an object which is attracting the patient's attention. The following movement and the reflex movements of the eyes can, however, be produced. In this group the lesion is to be localized below the cortex; probably the pathways descending from the frontal centers are injured not far above the nuclear region.

In a third group the paralyzed associated muscles react only to reflex stimulation; neither the voluntary and the attraction movements nor the following movement can be produced. In cases of paralysis of the lateral movements the internal rectus muscles are able to perform the convergence movement; in cases of paralysis of the vertical movement the elevator muscles may be able to function in Bell's phenomenon. In such cases the lesion must be localized close to the nuclei, the posterior longitudinal bundle being intact.

In a fourth group the paralyzed muscles respond neither to visual or other sensory stimuli nor to a word of command, nor are they able to perform a following movement or, finally, a reflex movement. In such cases either the posterior longitudinal bundle or the nuclei themselves must have been injured. The latter supposition is untenable in cases of paralysis of the lateral movements if the internal rectus muscles are able to produce a convergence movement and in cases of paralysis of the vertical movements if Bell's phenomenon is undisturbed.

In the last group of associated paralyses there are not only symptoms of a supranuclear lesion but also signs and symptoms indicating an injury of the nuclei, such as paralytic squint and diplopia and a variation in the mode of action of the paralyzed muscles according to the mode of stimulation.

To what I have said here about the supranuclear paralyses, I will add a few words concerning the disturbances observed occasionally in diseases of the extrapyramidal motor system and of the corpus striatum, respectively, for instance in paralysis agitans, pseudosclerosis, Wilson's disease and Huntington's chorea. Oculists observe these disturbances principally in patients who display the syndrome of so-called post-encephalitic parkinsonism, particularly the peculiar rigidity of the whole body, especially of the face so that it looks like a mask because of the lack of mimic movements and the paucity of lid and eye movements. Such cases may appear to be instances of real associated paralysis of the muscles of the eye and the patients must be carefully examined, since in encephalitis epidemica paralysis of individual muscles as well as of the associated movements may be encountered. The characteristics of the parkinson type present themselves in the following manner. The patient, apparently unable to move the eyes at a word of command, moves them spontaneously at times. Both the following movements and the reflex movements produced by sharp passive rotation of the head are perfect, although at times jerky. However, in contrast to the patient with true supranuclear paralysis, who cannot keep his eyes in the position attained by rotation of the head and who cannot prevent his eyes from returning to their original position as soon as the rotation ceases, the patient with paralysis of the Parkinson type, examined in the same way, can keep his eyes in the terminal position as long as his attention is directed to the point of fixation.

The cases of associated ocular paralysis caused by lesions within the cerebral hemispheres are mostly unsuitable for exact investigation, partly because of the bad mental condition of the patients and partly because of the quick recovery of ocular movements if the patient does not die. It may be supposed that an examination in some cases would reveal almost the same symptoms as in cases of pseudobulbar paralysis, which have already been discussed as the first group of associated ocular paralyses.

Conjugate deviation, which is found in most cases of recent cerebral lesion, may be caused by a variety of circumstances. If an associated pair of muscles is paralyzed in consequence of a lesion of the cortical center or of the path descending from it, the antagonistic pair of muscles at the same time ceases to receive its inhibitory innervation, because the excitatory and the inhibitory impulses to the agonists and the antagonists, respectively, are derived from the same center and from the same pathway, so that a lesion of one of them not only causes paralysis of the agonists but also prevents relaxation of the antagonists. Besides the loss of inhibitory impulses, a stimulation of the antagonists may be caused by a lesion which extends to the other hemisphere. The different causes of conjugate deviation make it easy to understand that the deviation in cases of cortical lesions is, as a rule, greater than in

cases of supranuclear paralysis of pontine origin and that it sometimes disappears within a few hours—as soon as the patient regains consciousness. Even if the conjugate deviation is essentially paralytic, it usually disappears rather quickly, probably because there are centers in both hemispheres for the parallel movement to the right and to the left, so that the center of the sound side soon begins to function vicariously. In the examples under discussion the patient's head is turned in the same direction as his eyes, that is to say, to the side of the lesion, except in those cases of patients with hemiplegia which are characterized by contracture of the limbs. In these cases the eyes and the head are turned toward the affected extremities.

Comparison of the Features of Conjugate Deviation in Cases of Lesions of the Hemispheres and in Cases of Pontine Lesions

Lesions of the Hemispheres	Pontine Lesions
1. In the first stage, regular and of considerable magnitude	1. Relatively rare and as a rule of small magnitude
2. Deviation usually of short duration	2. Deviation, if present, permanent
3. Deviation toward the side of the lesion	3. Deviation toward the opposite side
4. Deviation frequently a symptom of stimulation	4. Deviation usually a paralytic symptom, only rarely a stimulation symptom
5. Head turned (as a regular symptom) in the same direction as the deviation of the eyes	5. Abnormal position of the head not a typical symptom; if present, the head usually turned in the opposite direction to the deviation of the eyes
6. Associated paralysis of the muscles for contralateral movement, usually slight and transient	6. Associated paralysis in the direction of the lesion, nearly always severe and permanent
7. Invariably symmetrical functional disturbance of the associated muscles	7. Frequently asymmetrical paralysis of the associated muscles in consequence of the extension of the supranuclear lesion to the nucleus or the nuclei
8. Paralysis of the extremities and of the facial nerve collateral with the associated eye muscle paralysis	8. Paralysis of the extremities, if present, opposite to the side of the eye muscle paralysis; paralysis of the facial nerve, if present, usually collateral with the eye muscle paralysis
9. In lesions of both hemispheres all eye movements (including the vertical) restricted or impossible	9. In pontine lesions of both sides, paralysis of side-to-side movements without disturbance of the vertical movements

The accompanying table offers a comparison of the paralyses of lateral movements caused by pontine lesions and those caused by lesions in the hemispheres.

PARALYSES OF CONVERGENCE AND DIVERGENCE

Since it is known that the internal rectus muscles can be deprived of the ability to cooperate with the external rectus muscles in lateral movements without interference with their convergence function, it seems obvious at first sight that the latter function can also be lost without prejudice to the former. It is not known where the convergence center is located. In view of the fact that convergence paralyses are frequently caused by lesions within the region of the corpora quadrigemina, a subcortical convergence center is presumed to be in that

region; the isolated lesion of this center or of the pathway descending to the nuclei of both the internal rectus muscles must produce the symptoms of a pure convergence paralysis. In such a case the patient's eyes are unable to converge, whereas the internal rectus muscles function normally when cooperating with the external rectus muscles in lateral movements. There is crossed diplopia of near objects, whereas objects at a distance of more than 1 meter are seen single provided the convergence paralysis is not complicated by an exophoria. Even a slight degree of exophoria causes insuperable crossed diplopia of distant objects if the convergence is completely abolished. The angle of deviation is the same when the patient looks straight forward or to either side, but it is increased not only when he looks at near objects but also, as a rule, when he looks up and is decreased when he looks down, for the anatomic reasons previously mentioned. Variations of the signs and symptoms occur according to whether there is complete paralysis or only more or less considerable weakness of the convergence function.

True convergence paralyses of organic origin are rare. Many of the published cases are surely instances of functional disturbances, which are not always easy to distinguish from true paralyses. This difficulty is based on the exceptional position of the convergence movement among the ocular movements. Convergence does indeed belong to the fusion movements, but it is the only one which can be performed voluntarily. If binocular single vision is lost or has never existed, as in cases of strabismus acquired in early childhood, the ability to converge is more or less diminished or is absent, thus proving that the fusion faculty is the most essential factor in producing convergence, whereas the voluntary impulse to look at a near point is of only minor importance in bringing convergence into play. In spite of equally good vision in either eye, convergence is frequently defective or absent, especially in the myopic person who does close work without glasses. Since he sees distinctly at his punctum remotum, he avoids the fusion effort which is not assisted by the accommodation impulse and learns to suppress the retinal images of the nonfixing eye.

Lastly there occurs an insufficiency of convergence as a true functional neurosis not only in anemic and delicate persons or in patients convalescing after exhausting illnesses but also as a symptom of general neurasthenia or hysteria. Conditions of this type are sometimes wrongly diagnosed as convergence paralysis because convergence cannot be produced by means of the usual methods. No convergence is obtained if the patient is ordered to fix a near object, for instance his own finger or the nose or the fissure of Landolt's ophthalmodynamometer. But these tests do not offer reliable proof of the presence of a convergence paralysis unless it is ascertained that the patient has really been given the required impulse to look at the near object. This can be determined

by observing his accommodation and his pupils during the tests. Only if the pupils narrow and the refraction increases according to the distance of the near object can it be made certain that the deficiency of convergence is due to an organic lesion. I remember a patient who complained of a permanent crossed diplopia in near work after an accident. She was certified to be an invalid after several oculists had submitted a diagnosis of convergence paralysis of traumatic origin. When her eyes were tested in the usual manner there was no convergence. Since in the tests there was no accommodation and the pupils did not react I tried to ascertain whether the absence of cortical impulses was voluntary or involuntary. I asked the patient to tell me at which number the minute hand of my watch was pointing. Promptly she produced the corresponding convergence, accommodation, and pupillary action. I have seen several similar cases in which the patient at first seemed unable to converge but could be induced to do so by testing him with objects which attracted his attention and interest.

Another method suitable for deciding whether the lack of convergence is due to an organic lesion or is functional is the examination of the adduction power by means of prisms. In the case of convergence paralysis of organic origin crossed diplopia will arise as soon as an adducting prism is held before one eye. In the case of functional convergence paralysis binocular single vision will remain and the eye behind the rotary prism will move in accordance with the increasing strength of the prism until the limit of the adduction range is passed.

To make sure that the lack of convergence is a true paralysis of organic origin, the following conditions must be fulfilled: 1. There must be definite symptoms of an organic intracranial disease. 2. The convergence paralysis must have occurred rather suddenly. 3. The signs and symptoms tested at various times and by various methods must in a certain measure be constant. 4. The accommodation and the convergence reaction of the pupils must be producible without the corresponding convergence.

If internal ophthalmoplegia is found together with convergence paralysis, a lesion of the nuclear region and possibly also of the supranuclear pathway descending from the convergence center is certain.

DIVERGENCE PARALYSIS

Concerning divergence paralysis oculists are still at variance. Parinaud (1883) was the first to describe the signs and symptoms of divergence paralysis, and many authors have since confirmed his observations. Others, especially Berry and Alfred Graefe, have opposed the diagnosis, maintaining that the syndrome of so-called divergence paralysis should be explained in a different way.

The manifestations of a typical case of divergence paralysis include the following:

1. Homonymous diplopia, due to an abnormal convergence position, arises rather suddenly. It occurs for all objects beyond a certain distance, mostly beyond from 10 to 20 inches (25 to 50 cm.).

2. The angle of squint, being as a rule small or moderate, does not increase when the patient looks to the right or to the left; either it remains the same as in the primary position of the eyes or it even decreases. It increases or decreases, respectively, in looking down or up, according to the normal anatomic conditions formerly mentioned. This explains the habitual anomalous position of the head in some cases. The chin is depressed against the chest, so that by elevation of the visual lines convergence is transformed into parallelism, as has been demonstrated by photographs made in a similar case and discussed in my second lecture.[6]

3. When the fixed object is brought nearer to the patient, the two images approach each other and are finally fused when the object is from 10 to 15 inches distant. At this distance binocular single vision is maintaned in looking to the right or to the left as well.

4. When the object is brought still nearer, insufficient convergence causes diplopia.

5. Appropriate prisms, base out, give the patient binocular single vision, even of distant objects, in the whole field of fixation.

6. A restriction of the field of fixation is not found.

7. In repeated examinations at different times the angle of squint is found to be relatively constant.

Berry and Alfred Graefe denied the necessity of inferring from the syndrome just mentioned that there is a divergence paralysis. In their opinion these symptoms may be produced by a convergence spasm, and the validity of this argument must be admitted, as will be shown later. But it is not at all sufficient to disprove the occurrence of a divergence paralysis. In Berry's opinion there is no need to assume a divergence innervation, since the relaxation of the convergence innervation alone would make the visual lines return to parallelism. To this the reply can be made that a divergence innervation is required not only to change the convergence of the visual lines into parallelism but also to overcome an esophoria. Also, it is hardly conceivable that of all the innervations of the eye muscles the convergence innervation alone should lack an antagonistic innervation. Finally, tests for the range of duction have shown that there is an active divergence innervation which can be

6. Bielschowsky, A.: Lectures on Motor Anomalies of the Eyes: II. Paralysis of Individual Eye Muscles, Arch. Ophth. **13**:35 (Jan.) 1935.

increased to some extent by exercises. It would be wrong to assume that the limit of divergence is identical with the position of rest resulting from a complete relaxation of the convergence innervation. If this were so, every one with orthophoria would display a divergent squint on losing binocular single vision, whereas this occurs only in a certain percentage of patients.

So, from a theoretical point of view, the possibility of the occurrence of divergence paralysis must be conceded. On the other hand, I am convinced that in many cases a diagnosis of divergence paralysis is wrongly made. I have observed many patients with paralysis of one or both abducens nerves in whom typical symptoms were present at first but the characteristic symptoms were gradually lost and a concomitant type of deviation developed so that it no longer increased and decreased, respectively, in looking to the left and to the right. This transformation can sometimes occur even in a few days. If such a paralysis is seen only in the atypical stage, it is difficult to distinguish it from a divergence paralysis.

I have also observed patients with symptoms apparently typical of this kind of paralysis caused by a slight convergence spasm. I remember one patient with homonymous diplopia which had arisen four months previously. According to the report of his physician there had been intervals when no diplopia was present alternating with periods when the original diplopia was observed. The patient was extremely irritable and had been dismissed from military service on account of epileptic fits. In the first examination I found an abnormal convergence of 6 degrees when the patient's eyes were directed to a distant object. This slightly decreased when he looked to the left or to the right or up, increasing only when he looked down. When the object was brought closer, to a distance of 15 inches, binocular single vision was obtained in the whole field of fixation, whereas crossed diplopia arose for nearer objects. Prisms of 5 degrees placed base out before each eye also gave binocular single vision for distant objects. All of these symptoms coincided completely with those of typical divergence paralysis. Only repeated examinations brought discrepancies to light. When after the patient had closed his eyes for a fraction of a minute he opened them, crossed diplopia corresponding to a divergence of 2 degrees occurred, but only for a few seconds. Then the two images approached one another, fused and separated again, this time, however, as homonymous images, as a result of the gradual increasing of convergence up to 6 degrees. Such behavior is just as difficult to reconcile with divergence paralysis as is the fact that the patient, who initially required a prism of 5 degrees base out before each eye to obtain binocular single vision, was able to retain it even when the strength of the prisms was diminished. He was able to maintain it even when the prism had an

abducting effect, so that a divergence of 1 degree was produced. This lasted for only a few seconds, however, and then his eyes went back to the usual convergent position. Thus the true nature of the case was unmasked. It was not, as had been previously diagnosed, a case of divergence paralysis but one of slight convergence spasm occasionally met with in highly neurotic persons such as our patient. One could object to the diagnosis of convergence spasm since the patient displayed convergence insufficiency at reading distance, whereas in a case of spasm one would expect an increase of convergence. But it is by no means unusual to find an inability to transform convergence into parallelism in combination with a weakness of the convergence innervation—a peculiarity seen especially in neurasthenia, an important characteristic of which is the combination of increased irritability with abnormal exhaustibility.

A third anomaly could perhaps be mistaken for a divergence paralysis. If the fusion faculty is destroyed by a physical or a psychic shock, esophoria, which up to that time has been unnoticed, may become manifest and display symptoms similar to those of a divergence paralysis. As a rule, however, a thorough examination of the range of duction will show absence not only of divergence but also of other fusion movements. Although it must be admitted that the differential diagnosis is in many cases extremely difficult, sometimes even impossible, I am sure that the divergence innervation exists and that I have seen instances of a true divergence paralysis which not only presented the typical manifestations of the anomaly but—what is of decisive importance—also changed rather suddenly into an equally typical case of abducens nerve paralysis. Such a development is proof of an organic lesion, localized at first near the intact abducens nucleus but later extending and finally injuring the nucleus itself.

MUSCLE IMBALANCE IN MYOPIA

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The purpose of this communication is to report a short study of the prevalence of muscle imbalance in cases of myopia and its influence as a causative factor in the onset and progressiveness of this condition.

In an article published in 1895, I [1] laid stress on the fact that among the 245 cases of myopia there recorded not one could be described as simple myopia. At least one of three presumably congenital conditions —astigmatism, anisometropia and muscle imbalance—was demonstrable in each. In many cases all three were present. Experience since then has failed to demonstrate any case of myopia in which all these conditions could be positively excluded, and doubt therefore arose as to whether there is such a thing as simple myopia. If there is, it must be exceedingly rare. This refers, of course, only to axial myopia. In cases of myopia there are, therefore, two groups of conditions: (1) the congenital group, just referred to and (2) the pathologic elongation of the eyeball and the conditions secondary to it. It is assumed that the resistance of the scleral coat is subnormal, constituting what Poos called "that mysterious tendency towards myopia." and it is also assumed that the eye at birth is hypermetropic. It was found that, whereas 16 per cent of hypermetropic eyes were free from astigmatism, in the myopic group only 3.5 per cent were free. In the hypermetropic group unequal refraction existed in 70 per cent; in the myopic group it was present in 74 per cent. As regards muscle imbalance, it was found necessary to prescribe muscle correction in the myopic group in more than 50 per cent more cases than in other forms of refraction. Since myopia develops in eyes originally hypermetropic and is, therefore, always progressive in the early stage, the myopic process has at any rate a tendency to select eyes in which at least one of the three congenital anomalies mentioned is present. As there is no difficulty in determining the existence and measuring the degree of the refractive error, this article is written for the purpose of laying stress on the condition of the muscle balance.

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1. Marlow, F. W.: The Errors of Refraction in a Series of Two Thousand Eyes, New York M. J., 1895, vol. 62.

Two groups of 50 cases each are here presented for comparison: (1) cases in which no selection was made with regard to progressiveness or asthenopia and in which the patients were aged from 6 to 43 and (2) cases selected on account of (*a*) asthenopic symptoms or (*b*) progressiveness, in which an occlusion test was done for one of these reasons and in which the patients were aged from 7 to 62.

Table 1 gives the frequency of the phorias in the two groups. Of the cases in group 1, selected haphazard, orthophoria was present in 34 per cent. In the cases selected on account of progressiveness or asthenopic symptoms the percentage fell to 24 per cent. When the patients in the latter group of cases were subjected to a prolonged occlusion test orthophoria disappeared entirely. It is noteworthy that 70 per cent revealed hyperexophoria, a condition inducing excessive tension of all the muscles, both straight and oblique. Table 2 includes the cases in

TABLE 1.—*Frequency of Phorias in Two Groups Studied*

	Group 1	Group 2	
		Before Occlusion Test	After Occlusion Test
Orthophoria	17	12	0
Hyperphoria	10	10	4
Exophoria	10	7	6
Esophoria	4	2	0
Exophoria and hyperphoria	8	14	35
Esophoria and hyperphoria	1	5	5

the second group. The tentative conclusion from these statistics and from the experience of many years is that some fault in the muscle balance is almost always, if not invariably, present in cases of axial myopia. It is hard to resist the conclusion that a factor so constant in its occurrence must be an important element.

Its influence on the progressiveness of the disease cannot be conclusively established, mainly because of the common tendency for the process to become arrested after the period of growing is over. In other words, cessation of progress following the treatment of an imbalance might have taken place without it, owing to the usual tendency to stabilization with advancing years. Nevertheless, a case is occasionally seen in which it seems reasonable to credit correction of the imbalance with the result.

Some reference should be made to the diversity of opinion which exists as to the nature of some of the lower degrees of myopia, viz., whether they are developmental and physiologic, which Parsons and Suter think may be the case, or acquired and pathologic, which is the more commonly accepted opinion.

TABLE 2.—*Data for Group 2*

Case	Name, Age and Date	Age of Onset or First Glasses	Glasses Being Worn	Refraction	Phoria* Before Occlusion Test	Phoria* After Occlusion Test	Muscle Correction Added	Symptoms	Comment
1	W. F. O., aged 30, April 19, 1934	14 years (?)	—2.50 sph. for each eye	R, —2.50 sph. —0.25 cyl. ax. 10 L, —2.50 sph. —0.25 cyl. ax. 135	Ex, 4° L hyp, 3°	Ex, 28° L hyp, 3°	R, 4° in † L, 4¼° in, down 20°	Recent severe pain, agony since last glasses	No history of progress within last two years; relief
2	W. A. M., aged 51, Nov. 10, 1933	17 years	R, —1.50 sph. —0.75 cyl. ax. 110 L, —1.50 sph. —0.37 cyl. ax. 110	R, —1.75 sph. —0.87 cyl. ax. 105 L, —1.50 sph. —0.37 cyl. ax. 55	Ex, 0 L hyp, 1°	Ex, 4-6° Low L hyp	R, 2° in L, 2° in, down 12½°	Pain in neck, asthenopia, vertigo, gastric disorders	Symptoms due in part to dental abscess; relief
3	M. W., aged 62, Sept. 30, 1933	25 years (?)	R, —1.00 cyl. ax. 110 L, —1.75 cyl. ax. 180	R, —1.75 cyl. ax. 150 L, —1.25 cyl. ax. 25	Ex, 2° L hyp, 1°	Ex, 8° L hyp, ½°	R, 2½° in L, 2½° in, down 10°	Persistent asthenopia	Great relief
4	H. L. B., aged 16, April 19, 1934	10 years	R, —4.00 sph. L, —2.25 sph.	R, —4.00 sph. —0.25 cyl. ax. 55 L, —3.00 sph. —0.12 cyl. ax. 80	Ex, 2° L hyp, 1½°	Ex, 9° Low L hyp	R, 3° in L, 3° in, down 10°	Distant vision poor	Comfortable
5	V. C., aged 33, March 13, 1934	23 years	R, —1.50 sph. —2.50 cyl. ax. 180 ½° down L, —1.50 sph. —2.50 cyl. ax. 180	R, —1.50 sph. —3.00 cyl. ax. 2½ L, —1.75 sph. —3.00 cyl. ax. 175	Ex, 3° R hyp, 1½°	Ex, 10° L hyp, 3°	R, 2½° in, up 15° L, 3° in, down 35°	Headache, asthenopia, photophobia	Complete relief
6	S. J. M., aged 32, March 12, 1934	10 years	R, lost L, +0.25 sph. —0.75 cyl. ax. 180	R, —0.62 cyl. ax. 170 L, —0.62 cyl. ax. 5	Ex, 1°	Ex, 10° L hyp, ½-1½°	R, 2½° in L, 2¾° in, down 12½°	Constant headache	Complete relief
7	H. B., aged 31, Jan. 20, 1934	17 years	R, —1.00 sph. —1.50 cyl. ax. 180, prism 1° up L, —1.00 sph. —1.25 cyl. ax. 180	R, —1.00 sph. —1.50 cyl. ax. 175 L. —1.00 sph. —1.12 cyl. ax. 10	Ex, 0 L hyp, 3°	L hyp, 11-4° Ex, 6-4°	R, 3¼° in, up 55° L, 2½° in, down 55°	Asthenopia, foreign body sensation, photophobia, insomnia	Complete relief
8	W. E. V., aged 53, Jan. 4, 1934	In high school	R, +0.75 sph. —5.50 cyl. ax. 80 L, —1.12 sph. —1.25 cyl. ax. 130	R, +0.50 sph. —5.50 cyl. ax. 85 L, —0.75 sph. —2.00 cyl. ax. 135	Ex, 2° R hyp, ¾°	Ex, 4-6° R hyp, 2° +	R, 2½° in.† down 145° L, 2° in, up 165°	Headache, photophobia, asthenopia	"Never such relief"
9	V. W. P., aged 40, April 5, 1928	10 years	R, —0.50 sph. —0.25 cyl. ax. 15 L, —0.25 sph. —2.25 cyl. ax. 175	R, —1.00 sph. —2.00 cyl. ax. 17½ L, —0.75 sph. —2.25 cyl. ax. 17½	R hyp, ½°	Ex, 13° Hyp, varies, R and L	R, 3° in L, 3° in, down 15°	Headache, neckache, nervousness	Result unknown
10	R. A. Y., aged 31, April 2, 1929	?	0	R, —0.50 sph. L, —0.50 sph.	Ortho	Ex, 10° R hyp, 3°	R, 2½° in, down 160° L, 2½° in, up 165°	Gastric disorders, asthenopia	Relief
11	H. W. H., aged 34, October 1927	24 years	R, —0.25 sph. —0.50 cyl. ax. 90 L, —0.25 sph. —0.75 cyl. ax. 90	R, —1.25 sph. —1.00 cyl. ax. 115 L, —0.25 sph. —1.37 cyl. ax. 65 after scopolamine, 0.75 less myopic	L hyp, 1° Eso, 4°	L hyp, 2° Eso, 1°	R, 1½° up	Headache, strain	"Excellent satisfaction"

† The meaning in case 1 is: in the right eye, 4 degree prism base in, and in the left eye, 4¼ degree prism base in and down at 20 degrees. In case 8 the following is meant: in the right eye, 2½ degree prism base in and down at 145 degrees, and in the left eye, 2 degree prism base in and up at 165 degrees.

12	A. B. D., aged 46, January 1928	16 years	—0.50 sph. —0.75 cyl. ax. 180 in each eye	R, —0.50 sph. —0.62 cyl. ax. 5 L, —1.00 sph. —0.25 cyl. ax. 175	L hyp, 1°	Ex, 6° L hyp, 2-3°	R, 2½° in, up 45° L, 2° in	Irritable, indigestion	Complete relief
13	J. B., aged 47, March 1929	43 years	—0.50 cyl. ax. 180 for each eye	R, —0.25 sph. —0.12 cyl. ax. 175 L, —0.25 sph. —0.12 cyl. ax. 15	L hyp, 1-2°	Ex, 5½° R hyp, 1°	R, 2° in down 155° L, 1¾° in	Cannot get glasses to fit, photophobia, asthenopia	Complete relief
14	A. A. M., aged 24, Feb. 20, 1929	22 years	R, —1.25 sph. —0.25 cyl. ax. 95 L, —1.25 sph. —0.25 cyl. ax. 100	R, —1.50 sph. —0.25 cyl. ax. 100 L, —1.50 sph. —0.25 cyl. ax. 75	Ex, 1°	Eso, 6° L hyp, 1°	R, 1¾° out, up 155° L, 1¾° out	Drawing, blur, asthenopia	Relief after 6 months
15	C. K., aged 21, September 1925	9 years	R, —3.00 sph. L, —3.50 sph.	R, —2.50 sph. —0.75 cyl. ax. 180 L, —4.50 sph. —0.25 cyl. ax. 20	Ex, 1° Hyp, 0	Ex, 5° L hyp, 1¾°	R, 1½° in, L, 1¾° in, down 35°	Distant vision blurred, headache, sleepy	Complete relief
16	S. C. C., aged 42, March 8, 1928	In college	R, —2.25 sph. —0.25 cyl. ax. 90 L, —1.75 sph. —0.25 cyl. ax. 90	R, —2.25 sph. —0.25 cyl. ax. 120 L, —2.00 sph. —0.25 cyl. ax. 60	R hyp, 1°—	Ex, 12° R hyp, 2½°	R, 3¼° in, down 160° L, 3° in	Asthenopia, failure to get relief	Complete relief
17	R. A. B., aged 25, May 15, 1925	?	0	R, —0.25 sph. —0.12 cyl. ax. 45 L, —0.25 sph. —0.12 cyl. ax. 105	Ortho	L hyp, 2°	R, 1° up L, ½° down	"Terrible headache"	Complete relief
18	W. S., aged 35, April 1, 1926	25 years	R, —0.87 sph. —0.62 cyl. ax. 15 L, —0.62 sph. —1.50 cyl. ax. 180	R, —1.00 sph. —1.00 cyl. ax. 20 L, —0.62 sph. —1.50 cyl. ax. 180	Ex, 1° L hyp, ¾°	L hyp, 2½° Ex, 8½°	R, 2¾° in, up 40° L, 2° in	Headache, asthenopia, photophobia	Myopia diminished; 1931: R, —0.50 —1.00 cyl. ax. 25; L, —0.25 —2.00 cyl. ax. 5
19	G. M., aged 32, July 1927	10 years	R, —0.50 cyl. ax. 110 L, —0.70 cyl. ax. 60	R, —0.25 sph. —1.25 cyl. ax. 115 L, —0.25 sph. —1.00 cyl. ax. 70	Ex, 1°— Hyp, 0	Ex, 6° L hyp, 1½°	R, 2° in, up 25° L, 1¾° in	Extreme asthenopia for several years	Complete relief
20	J. F. H., aged 35, July 1927	In high school	? None now	R, —0.25 sph. L, —0.25 sph. —0.25 cyl. ax. 30	Ortho	Ex, 8° Hyp, 0	R, 2½° in L, 2½° in	Photophobia, indigestion, headache	Complete relief
21	P. B. N., aged 54, July 8, 1927	16 years	R, +0.50 sph. —2.25 cyl. ax. 180 1° up L, —1.75 cyl. ax. 5	R, +0.75 sph. —2.50 cyl. ax. 180 L, —2.00 cyl. ax. 20	L hyp, 2°	Ex, 2° L hyp, 4°	R, ¾° up L, down 1½°	Contraction in neck, indigestion	Complete relief
22	A. C. W., aged 19, May 1908	12 years or earlier	?	R, —1.50 sph. L, —1.25 cyl. ax. —0.37 cyl. ax. 20	Ortho	L hyp, 2° Ex, 11°	R, 3½° in L, 3½° in, down 150°	Headache	No record
23	G. L., aged 32, March 7, 1934	15 years	R, —1.75 sph. —0.50 cyl. ax. 90 L, —1.75 sph. —0.50 cyl. ax. 90	R, —1.75 sph. —0.62 cyl. ax. 100 L, —1.75 sph. —0.62 cyl. ax. 90	R hyp, ¾°	R hyp, 2° Ex, 7°	R, 3° in, down 12° L, 2° in, down 25°	Headache, photophobia	Result unknown
24	W. N., aged 32, May 26, 1925	For near work only	R, —0.37 sph. —0.37 cyl. ax. 125 L, —0.37 sph. —0.25 cyl. ax. 170	R, —0.37 sph. —0.37 cyl. ax. 140 L, —0.12 sph. —0.62 cyl. ax. 170	R hyp, ½°	L hyp, ½°— Ex, 6°	R, 1¾° in L, 1¾° in	Headache	No relief until after hysterectomy

* Ex indicates exophoria; hyp, hyperphoria; eso, esophoria, and ortho, orthophoria.

TABLE 2.—*Data for Group 2—Continued*

Case	Name, Age and Date	Age of Onset or First Glasses	Glasses Being Worn	Refraction	Phoria*		Muscle Correction Added	Symptoms	Comment
					Before Occlusion Test	After Occlusion Test			
25	K. N., aged 31, Oct. 5, 1927	23-24 years temporarily	None now	R, —0.25 sph. —0.25 cyl. ax. 50 L, —0.25 sph. —0.25 cyl. ax. 110	Ex, 1°	Ex, 8° L hyp, 3½°	R, 2¾° in, up 20° L, 3° in, down 30°	Pressure in head, nervousness	Temporary relief
26	A. M. M., aged 48, March 19, 1926	?	For near work only	R, —0.25 sph. —0.50 cyl. ax. 140 L, —0.25 sph. —0.25 cyl. ax. 70	Eso, 2° R hyp, ¾°	Low eso R hyp, 1°	R, ¾° down	Headache, photophobia	Result unknown
27	W. B. M., aged 48, March 17, 1920	34 years	R, —0.87 cyl. ax. 80 L, —0.87 cyl. ax. 10	R, —0.50 cyl. ax. 175 L, —0.75 cyl. ax. 5	Ortho	R hyp, 4°	R, ¾° down L, 1½° up	Headache, nervousness	Great relief
28	E. W., aged 7½, March 13, 1919	Recent	0	R, —0.50 sph. L, —0.25 sph.	Ortho	L hyp, 5° Ex, 5°	R, 3° in, up 45° L, 3½° in, down 40°	Asthenopia and progressiveness	Relief case 3 (fig. 3)
29	E. A., aged 7, Oct. 27, 1917	Recent	0	R, —0.25 sph. —1.50 cyl. ax. 180 L, —4.50 sph.	Ortho	Ex, 7° Low R hyp	R, 2° in L, 2° in, up 150°	Distant vision poor	Case 1, figure 1
30	M. S., aged 47, Feb. 27, 1925	13 years	R, —3.25 sph. —0.75 cyl. ax. 160 L, —4.00 sph. —0.25 cyl. ax. 170	R, —4.00 sph. —0.50 cyl. ax. 165 L, —4.00 sph. —0.25 cyl. ax. 145	L hyp, 1°	L hyp, 1° Ex, 7½°	R, 2½° in L, 2½° in, down 10°	Intense sick headache, photophobia	"A new person"
31	E. G., aged 9, January 1913	Recent	0	R, —0.75 sph. L, —0.87 sph.	R hyp, ½°	L hyp, 2° Ex, 6°	R, 2° in L, 2° in, down 20°	Headache	Relief
32	W. Y., aged 22, Dec. 8, 1924	19 years	?	R, —0.25 cyl. ax. 75 L, —0.25 sph. —0.25 cyl. ax. 135	Ex, 1° L hyp, 1°—	Ex, 2° L hyp, 1°—	R, 3° in L, 3° in, down 15°	Terrible headaches, nausea, vomiting	Relief; how long ?
33	J. K., aged 14, December 1932	Recent	0	R, —1.00 sph. L, —0.50 sph. March 1934: R, —2.00 sph. L, —1.25 sph.	Ortho	Ex, 10° R hyp, 3°	R, 2½° in, down 25° L, 3° in, up 40°	Progressive myopia	Comfortable
34	L. B., aged 12, January 1920	12 years	0	R, —2.25 sph. L, —2.00 sph.	R hyp, 2°	Ex, 8-9° R hyp, 4° (at age of 16)	R, 3° in, 150° down L, 3° in, up 15°	Headache, progressive myopia	Slowing down of myopia
35	B. J. T., aged 50, July 1924	42 years	R, —2.50 cyl. ax. 170 L, —2.50 cyl. ax. 5	R, —3.25 cyl. ax. 170 L, +0.50 sph. —3.50 cyl. ax. 10	Eso, ¾° Hyp, 0	Eso, 1° L hyp, 1½°	L, 1° down	Constant headache	Complete relief
36	F. R., aged 32, July 1924	27 years	R, —0.50 sph. +0.37 cyl. ax. 120 L, —0.25 cyl. ax. 150	R, —0.87 cyl. ax. 15 L, —0.25 sph.	Ortho	Ex, 2° R hyp, 2°	R, ¾° in L, 1¾° in, up 130°	Headache, photophobia	Relief
37	E. H., aged 13, July 1924	.....	—	R, —0.25 sph. —0.25 cyl. ax. 30 L, —0.25 sph.	Ex, 1°	Ex, 13° L hyp, 4°	R, 2½° in, up 40° L, 2½° in, down 20°	Headache, styes	Complete relief

38	A. P., aged 16, September 1921	16 years	0	R, —0.25 sph. +0.50 cyl. ax. 90 L, —0.25 +0.50 cyl. ax. 90	Eso, 1°; 1926, eso, 1°	Ex, 12 R, —0.75 —0.37 cyl. ax. 10 L, —0.75 —0.62 cyl. ax. 160	2½° in for each eye	Progressive myopia, asthenopia	1930: progress stopped
39	M. H., aged 21, October 1927	13 years	—3.00 sph. for each eye	—3.00 sph. each	Eso, 3° L hyp, ¾°	Ex, 4½°	2° in for each eye	Nearsighted, headache	Result unknown
40	A. S., aged 27, March 1927	21 years; near only	R, —0.37 sph. —0.12 cyl. ax. 180 L, —0.25 sph.	R, —0.50 sph. —0.37 cyl. ax. 165 L, —0.25 sph. —0.25 cyl. ax. 5	Ex, 1° L hyp, 1°	Ex, 3° L hyp, 2½°	R, 1¾° in, up 55° L, 1° in	Asthenopia	Result unknown
41	L. M., aged 10, January 1926	6 years	R, —0.25 —0.75 cyl. ax. 100 L, —0.25 —0.75 cyl. ax. 60	R, —0.75 cyl. ax. 110 L, —0.50 sph. —0.75 cyl. ax. 55	Ex, 1½° R hyp, ¾°	L hyp, 2°	L, 1½° down	Asthenopia, headache	Complete relief
42	A. J. M., aged 30, May 1926	0	?	R, —1.00 sph. L, —0.75 sph.	Ex, 5° L hyp, ½°—	L hyp, 2½° Ex, 7°	R, 2° in, up 15° L, 2¼° in, down 25°	Headache; took one year to relieve completely	Complete relief finally
43	A. G., aged 19, September 1925	9 years	—2.75 sph. for each eye	R, —2.75 sph. —0.25 cyl. ax. 65 L, —3.00 sph. —0.50 cyl. ax. 55	Ex, 1° R hyp, ½°	Ex, 12° Hyp, 0	3° in for each eye	Headache	Partial relief
44	Miss M., aged 22, September 1926	14 years	R, —1.00 sph. L, —0.75 sph. —0.25 cyl. ax. 90	R, —0.75 sph. —0.25 cyl. ax. 45 L, —0.75 sph. —0.12 cyl. ax. 70	Ex, 1°	Ex, 5° L hyp, 2¼°	R, 1½° in L, 2¼° in, down 50°	Terrible headache, photophobia	Relief
45	A. E. J., aged 35, February 1926	20 years	R, —3.00 sph. —0.37 cyl. ax. 50 L, —2.00 sph. —0.37 cyl. ax. 160	R, —3.00 sph. —0.37 cyl. ax. 50 L, —2.00 sph. —0.37 cyl. ax. 165	Ortho	Ex, 3°	1° in for each eye	Severe sick headache	Relief
46	Mrs. B., aged 36, March 1928	14 years	0 (?)	R, —2.00 sph. —0.12 cyl. ax. 175 L, —2.50 sph. —0.12 cyl. ax. 5	Ex, 3° L hyp, ½°	Ex, 10° L hyp, 1½°	R, 2¼° in L, 2½° in, down 15°	Severe headache	Complete relief
47	A. B., aged 32, November 1920	?	0	R, —0.37 cyl. ax. 160 L, +0.50 sph. —1.25 cyl. ax. 180	Ex, 2° L hyp, ½°	Ex, 16°	3° in for each eye	Insomnia, twitching, headache	Complete relief finally
48	Mr. S., aged 40, December 1925	12 years	R, —3.25 sph. —1.50 cyl. ax. 180 L, —2.50 sph. —1.75 cyl. ax. 170	R, —3.50 sph. —0.87 cyl. ax. 7½ L, —2.50 sph. —2.50 cyl. ax. 175	Eso, 1° L hyp, 2°	Ex, 5° L hyp, ½°	R, 1½° in L, 1¾° in, down 15°	Headache, asthenopia	
49	S. A. W., aged 36, October 1930	19 years	..... Scopolamine hydrobromide R, —1.00 sph. —0.25 cyl. ax. 90 L, —1.25 sph. —0.25 cyl. ax. 60	R, —0.25 sph. —0.25 cyl. ax. 90 L, —0.50 sph. —0.50 cyl. ax. 40	Ortho	Ex, 4½° L hyp, 3°	R, 2° in L, 2¼° down	Neurasthenia	Complete relief
50	J. S., aged 29, April 1924	?	0	R, —0.50 cyl. ax. 160 L, —0.25 sph.	Eso, 2°	Eso, 2° R hyp, 5½°	R, 2¾° down, out 65° L, 2½° up, out 55°	Profound neurasthenia, photophobia, headache, vertigo	Complete relief

* Ex indicates exophoria; hyp, hyperphoria; eso, esophoria, and ortho, orthophoria.

Risley [2] argued strongly against the developmental view and stated:

> It must be shown that the increase in the refraction of the eye is consonant with the health of the organ, and that it is conducive to greater comfort and usefulness to the individual, before we can accept the view which regards the myopic eye as a manifestation of normal growth, or of "an appropriate adaptation." If in either or both of these respects demonstration is wanting, or if the contrary should appear on sufficient investigation, then the increasing percentage of nearsight in the community must be classed among the baneful physical results of our civilization.

If some of the cases are developmental and therefore finally stationary, how can they be differentiated from ordinary axial myopia?

Is the myopia congenital?

Can myopia beginning several years after birth be regarded as developmental?

There is also great diversity of opinion as to the age of the onset of myopia. Thus, Ernest Clarke stated that it is "computed" that about 1 in 1,000 children under 5 years of age is myopic. Fuchs believed that about 5 per cent of infants are myopic. Swanzy gave the period of onset as between 8 and 15 years, infants being hypermetropic. Weeks stated that up to 8 years of age from 3 to 5 per cent of children are myopic.

In making out variation frequency curves of refractive errors (Gauss), some authors think that only developmental cases should be included and therefore that all cases showing changes of the fundus should be excluded. Others (for example, Tron) believe that all cases in which myopia is greater than 6:00 D. should be excluded. Dunstan would include all cases in which vision is normal with correction. As the line of demarcation evidently is vague and arbitrary, it seems necessary to avoid the attempt to draw it and for the present to regard all cases as of the same nature.

If all hypermetropic and myopic variations are admitted in constructing such a curve, an unsymmetrical or skewed curve results, which is not surprising. On one side of the peak the variations are physiologic and on the other pathologic. Such a curve is useful for comparison or contrast only.

Certain factors meet with more or less general acceptance: an undue plasticity or softness of the sclera, necessarily diminishing with advancing age and maturity, and, in addition, the previously mentioned congenital conditions, of which one or more will invariably be found. Astigmatism was present in 96.5 per cent of the original 245 cases studied and in 88 per cent of the group recently studied. Anisometropia was found in 74 per cent of the original 245 cases and in 72 per cent

2. Risley, S. D., in Norris, W. F., and Oliver, C. A.: System of Diseases of the Eye, Philadelphia, J. B. Lippincott Company, 1897-1898.

of the group recently studied, while heterophoria was present in 48 per cent of the original group and in all the cases recently studied in which the occlusion test was carried out.

In view of the fact that myopia of even high degree is found among those who never use their eyes for near work, Fuchs stated that "some therefore argue that there is some congenital basis for myopia which will be effective in causing its progressive increase whether the eyes are used for *near* work or not." May not the pressure exerted *constantly* on an abnormally plastic sclera by tense extrinsic muscles in the interest of fusion for *all* distances supply the basis?

The tendency to fuse for the purpose of binocular single vision no doubt antedates the desire for clear vision. To see an object in mass, as one, must precede the attempt or instinct to make out its details. In other words, while astigmatism has admittedly an important influence in increasing myopia after use of the eyes for defining the nature or details of objects has become desirable or instinctive, muscle coordination begins at a much earlier age and therefore, by acting on an abnormally soft eye, may be responsible for the onset as well as the progressiveness of the myopia. Professor Watson has shown that the "eye hand" association begins between the one hundred and thirtieth and one hundred and fiftieth day of life, and Worth has shown that infants between 12 and 18 months of age readily make a compensating movement for a prism with the apex in. He affirmed, moreover, that in infants between 5 and 6 months of age one finds the first certain evidence of a desire for binocular vision, though probably a certain degree of binocular vision is present at a much earlier period. It seems reasonable to assume that the desire and necessity for clear definition of small objects must arise at a much later period, particularly with training in observation at the beginning of the school period.

Ernest Clarke has clearly shown that the theory that the increase of myopia is due to the increased convergence due to abnormally great interpupillary distance is without foundation. Also, in association with Sir Arthur Keith, he studied the theory that the shape of the orbit is a causative factor. A comparison of a large series of skulls of all races makes this theory untenable. In the paper of Ernest Clarke, read at the convention of English-speaking ophthalmologic societies in 1925, the influence of astigmatism was clearly recognized. It has also been noted that Risley [2] mentioned cases of hypermetropia passing on to myopia "through the turnstile of astigmatism." Every ophthalmologist of experience must have seen this happen. No reference was made in either of these articles to the possible influence of muscle imbalance, certainly not of latent muscle imbalance, which can be revealed only by prolonged occlusion.

It is easy to see how astigmatism by lowering vision tends to cause reading and other work to be done at a shorter distance and therefore under a larger visual angle and so to increase convergence, but it is a matter of common observation that many myopes, at any rate at an early stage, do not read at an unduly near point, and consequently the influence of astigmatism by causing increased convergence may be ruled out in such cases. On the other hand, the tension due to automatic efforts to offset muscle imbalance is constant and inescapable.

It can scarcely be doubted that the constant excessive pressure by the extrinsic muscles necessary to maintain fusion, acting on a sclera of subnormal resistance, is one of the factors causing myopia to progress. The pressure so exerted is of the same nature, though of less degree, as that inherent in convergence for the near point, especially for too near a point, but, being constant, may be of equal or perhaps of more importance. Accepting the theory that axial myopia is due to pressure of tense muscles acting on a weak sclera, Luedde has shown convincingly the importance of diminishing the convergence by monocular cycloplegia.

Pollock of Glasgow [3] also showed some years ago that atropine instilled into both eyes for a long period brought about a remarkable reduction in the degree of myopia in some cases.

May not part of the benefit shown by Luedde and Pollock to follow this procedure be due to the diminished stimulus to fusion (blurred retinal images), not only for the near point but also for all distances? It must be noted that monocular cycloplegia induces or permits relaxation of that part of the near point convergence only which offsets the near point phoria. The part associated with accommodation is unaffected.

REPORT OF CASES

The following cases, in which observations on the refraction and muscle balance covering a number of years have been made, are of interest as having some bearing on the subject.

CASE 1 (chart 1).—E. A., when first seen at the age of 7, had myopia of 4.50 D. in the left eye and 0.50 D. in the right. There was astigmatism of about 1.25 D. in each eye; this varied little during the whole period she was under observation, which was from the age of 7 to the age of 21. Between the ages of 7 and 12 there was a great increase in the myopia of the right eye, which now amounted to 3.50 D., and also an increase in the myopia of the left eye, now amounting to 5.50 D. An occlusion test was then done, which showed exophoria of 6 or 7 degrees and practically no hyperphoria. For four years following the correction of the exophoria, that is, from 12 to 16 years of age, there was only a slight increase in the myopia, which was 1.00 D. in the right eye and 0.50 D. in the left. After that there was an increase in the myopia, which finally amounted to 7.50 D. in the left eye and 6.00 D. in the right. In this case the correction

3. Pollock, W. B. I.: Myopia in Children, Glasgow M. J. **86**:214, 1916.

of the muscle error was followed by a slowing down of the myopic process for the following four years. Several members of the patient's family were myopic.

CASE 2 (chart 2).—A. P. was first seen at the age of 16, when he complained of aching. Vision was normal, but there was a little hypermetropic astigmatism.

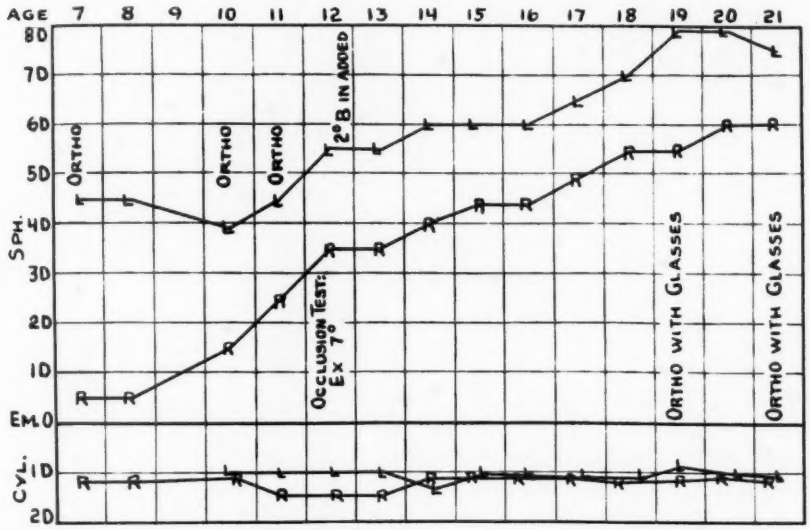

Chart 1 (case 1).—Graph showing slowing down of myopic process following correction of muscle imbalance. In the charts the following abbreviations are used: *Ex* indicates exophoria; *Hyp*, hyperphoria; *Ortho*, orthophoria, and *Eso*, esophoria. *Em* indicates emmetropia, and *B* indicates base.

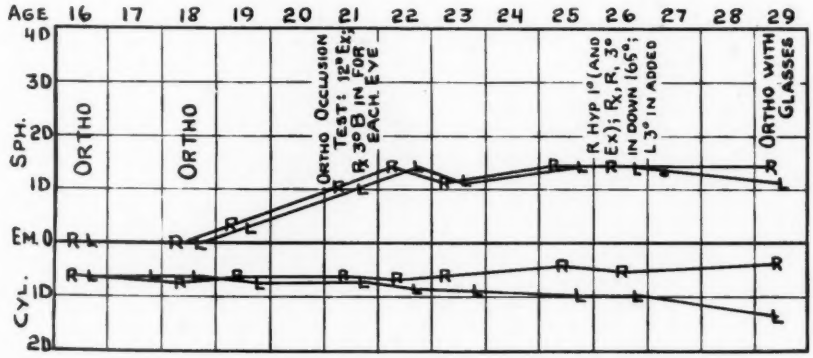
AGE 16 17 18 19 20 21 22 23 24 25 26 27 28 29
SPH.
CYL.
EM.
ORTHO
ORTHO
ORTHO OCCLUSION TEST: 12° EX; R 3° B IN FOR EACH EYE
R HYP 1° (AND 8° EX); R, R, 3° IN DOWN 165°; L 3° IN ADDED
ORTHO WITH GLASSES

Chart 2 (case 2).—Graph showing arrest of myopia following correction of muscle imbalance.

Two years later he complained of headache and blurring of distant vision; the right eye was now accepting a —0.62 cyl., and the left eye a —0.50 cyl. Muscle tests showed orthophoria. One year later the eyes were slightly more myopic (—0.50 D.). Two years later, at the age of 21, tests under scopolamine hydrobromide showed myopia of 1.00 D. in each eye, astigmatism as was before noted, and orthophoria. As the asthenopic symptoms were still present, an occlu-

sion test was done, which revealed 12 degrees of exophoria; 2½ degrees base in for each eye was prescribed, with full correction of the myopia and astigmatism. Two years later, when the patient was 23 years old, he showed an increase of myopia of 0.50 D. At the age of 29, when he was last seen, the myopia had remained stationary in the right eye and was slightly less in the left. It appears that the correction of the muscle imbalance in this case had a marked influence in arresting the progress of the myopia. The asthenopic symptoms were not relieved until after the imbalance was allowed for.

Case 3 (chart 3).—E. W. was first seen at the age of 7, when she complained of difficulty in seeing the blackboard and tiring of her eyes. The right eye accepted a —0.50 sph. and the left eye a —0.25 sph. after the instillation of scopolamine hydrobromide. The patient had vision of 6/6— in each eye. At this time tests showed a muscle balance varying from orthophoria to exophoria of 4 degrees. At the age of 9 vision and refraction remained unchanged, but the patient was suffering a great deal from headache and complained again of trouble in seeing the blackboard. Examination under scopolamine hydrobromide gave the same results as previously. Later in this year she complained again of trouble in seeing,

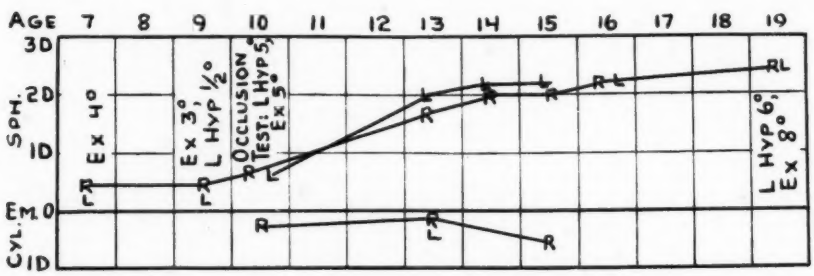

Chart 3 (case 3).—Graph showing stationary tendency of myopia after correction of muscle error.

with much headache and irritability. An occlusion test was then done for seven days, which showed 5 degrees of left hyperphoria and 5 of exophoria. The symptoms were greatly relieved by partial correction of this error. At the age of 10 she saw best with a —0.75 sph. for each eye. When she was 13 years old there was a complaint of difficulty in seeing in the distance. She showed myopia of 1.75 D. in the right eye and 2.00 D. in the left, with low degrees of astigmatism. From that date until she was 21 years old there was almost no change in the myopia. She accepted a —0.50 sph. in addition to the previous correction. It may be seen from chart 3 that from the tenth to the thirteenth year there was an increase in the myopia of about 1.25 D. and that after that date the myopia remained practically stationary.

It is difficult to say how much influence the correction of the muscle error had on the progress of the myopia. Progressiveness evidently started about the time that the occlusion test was done. Also, as the patient was not seen between the ages of 10 and 13 years, it is impossible to say just when it ceased to progress after the occlusion test was done. The case is of interest in exemplifying how a high degree of muscle imbalance may be present in a case of apparently simple myopia. It is hard to say how much influence the correction of the imbalance had in stabilizing the myopia.

CASE 4 (chart 4).—A. S. was seen first at the age of 12, when she showed myopia of 2.25 D. in the right eye and 1.75 D. in the left. She was seen again at the age of 16; the myopia of the right eye had increased to 5.50 D. and that of the left eye to 4.00 D. An occlusion test was then done, which revealed 4 degrees of right hyperphoria and 9 degrees of exophoria. Following the correction of these errors, the myopia of the right eye went down to 5.00 D., that of the left being unchanged. From that date until she was 24 years of age there was no increase in the myopia, that of the left eye remaining the same and that of the right being the same as when she was 18 years of age, there having been an intervening period during which the myopia of the right eye was 5.00 D. A

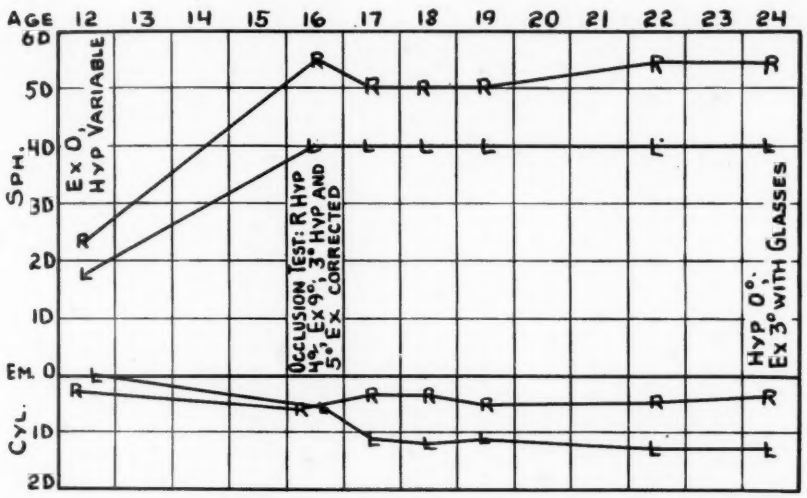

Chart 4 (case 4).—Graph showing progress of myopia checked following correction of muscle imbalance.

glance at the chart (chart 4) suggests that the correction of the muscle imbalance had a marked influence in controlling the progress of the myopia.

CASE 5 (chart 5).—E. G. was first seen on Jan. 16, 1913, at the age of 9, on account of defective distant vision. She had worn no glasses hitherto. Vision was 6/36 in each eye; after the instillation of scopolamine hydrobromide she showed myopia of 0.75 D., no astigmatism and vision of 6/6. In December 1914 under scopolamine hydrobromide, she accepted a —4.00 sph. —0.25 cyl. ax. 50 for the right eye and a —3.50 sph. —0.50 cyl. ax. 115 for the left. In 1919, refraction was practically unchanged, but on account of persistent headache an occlusion test was done, which showed 2 degrees of left hyperphoria and 6 degrees of exophoria. A correction of 4 degrees of exophoria and 1¼ degrees of hyperphoria was added to the refractive correction. Since then she has been comfortable, and there has been no increase in the myopia. The myopia remained unchanged for six months before the occlusion test was done, and it is noteworthy that no change took place after it.

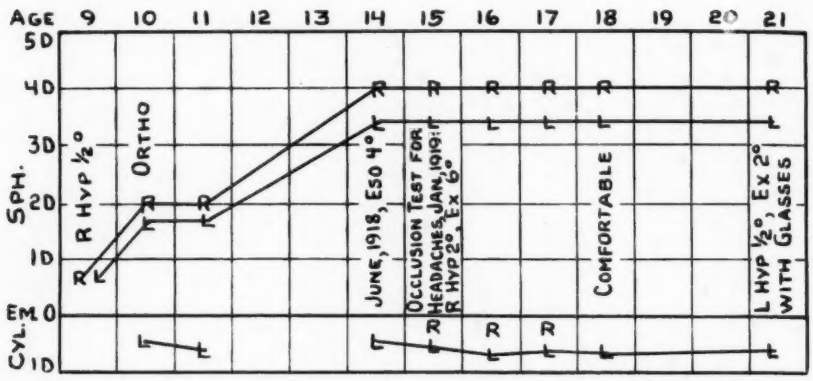
AGE 9 10 11 12 13 14 15 16 17 18 19 20 21
SPH.
CYL.
EM.
R HYP ½°
ORTHO
JUNE, 1918, ESO 4°
OCCLUSION TEST FOR HEADACHES, JAN. 1919: R HYP 2°, EX 6°
COMFORTABLE
L HYP ½°, EX 2° WITH GLASSES

Chart 5 (case 5).—Graph showing effect on myopia of correction of muscle error.

Occasionally cases have been seen in which there has been a diminution of the myopia following correction of the muscle balance:

CASE 6 (chart 6).—J. E. R., a medical student, aged 22, whose father and grandfather had progressive myopia, had worn glasses since the age of 17. The glasses being worn when he was seen in March 1926, when he was 22 years old, were —2.00 sph. —0.50 cyl. ax. 180 for the right eye and —2.50 sph. for the

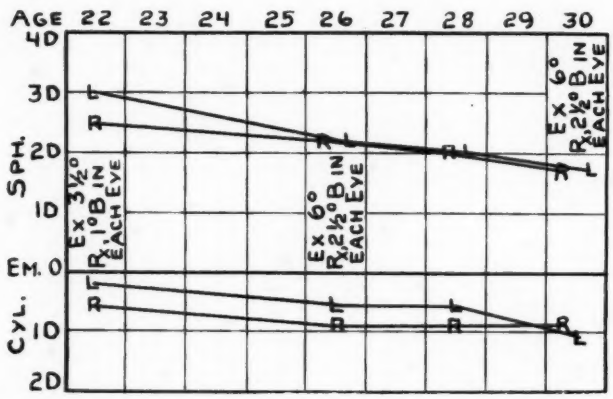

Chart 6 (case 6).—Graph showing recession of myopic process following correction of muscle imbalance.

left, without muscle correction. At examination on this date he accepted a correction of 2.50 —0.50 cyl. ax. 180 for the right eye and —3.00 —0.25 cyl. ax. 5 for the left. In other words, there was an apparent increase in the myopia within the two year period preceding this examination. It was found that he had an exophoria of 3½ degrees, 2 degrees of which were corrected by the new prescription.

He was seen again in January 1930, when he accepted a correction of —2.50 —0.87 cyl. ax. 175 for the right eye and —2.50 —0.62 cyl. ax. 175 for the left. On this date exophoria of 6 degrees could be demonstrated; 5 degrees of the exophoria were now corrected.

In June 1932, he accepted a correction of —2.00 in each eye, with the astigmatic correction already described and now showed 9 degrees of exophoria.

In June 1934, the spherical correction required was —1.75 in each eye, with the cylinders already described. In other words, there was brought about marked recession of the myopia following correction of the exophoria.

No cycloplegic was used in this case, nor was an occlusion test done, and therefore the improvement may have been due to relaxation of an accommodative spasm following the muscle correction. This is not an uncommon occurrence, as was pointed out in an article in the ARCHIVES.[4] While it is well known that excessive accommodative effort may induce esophoria or esotropia, it is not commonly realized that an excessive convergence effort may induce an accommodative spasm. A striking case is recorded here. Whichever is the correct explanation, the importance of muscle correction is apparent.

CONCLUSIONS

1. Muscle imbalance is present in a high percentage of cases of axial myopia, if not in all.

2. It is an important, probably the most important, active factor in the onset and progress of this condition.

3. In view of the high degree of latency of muscle imbalance, an occlusion test should be systematically carried out in all cases at as early a stage as possible.

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4. Marlow, F. W.: Persistent Accommodative Spasm Due to Latent Hyperphoria, Arch. Ophth. **51**:223 (May) 1922.

SALZMANN'S NODULAR CORNEAL DYSTROPHY

ITS PATHOLOGIC PROCESS AND A SUGGESTED THERAPY

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AND
DEWEY KATZ, M.D.
CHICAGO

Salzmann's nodular corneal dystrophy differs from Groenouw's nodular corneal dystrophy in that the dystrophic nodules are larger and less numerous, the elevation of the nodules above the surface of the cornea is more pronounced, the disease appears in all decades of life, it predominantly affects the female sex, and it is not familial. The majority of the cases were unilateral at the time of their description, and almost always the diseased eyes showed evidence of repeated attacks of eczematous keratoconjunctivitis either in the form of the usual corneal opacities or in the form of eczematous pannus.

E. Fuchs [1] also had observed the development of raised corneal areas in cases of eczematous keratoconjunctivitis, for in an article published in 1902, he stated:

> At times the areas of nodular opacity above the corneal surface resemble certain cases of old eczematous keratitis in which one or more of the persisting lesions takes on a thickening of the epithelium, possibly also a keloid formation of the underlying scar, so that small bluish-white elevations of the corneal surface arise.

Salzmann was of the opinion that case 8 in Fuch's article belonged to the variety of nodular corneal dystrophy which he described. Except for this one case, Salzmann, in 1925, was not aware of any report or description of a case or cases which belonged to this clinical entity.

In his [2] original publication on this subject in 1925, Salzmann stated that in view of the relatively large number of such cases (twenty-three)

From the Division of Ophthalmology, University of Chicago, E. V. L. Brown, director.

Read by invitation at the meeting of the American Ophthalmological Society, July 9, 1934.

1. Fuchs, E.: Ueber knötchenförmige Hornhauttrübung, Arch. f. Ophth. **53**:423, 1902.

2. Salzmann, M.: Ueber eine Abart der Knötchenförmigen Hornhautdystrophie, Ztschr. f. Augenh. **75**:92, 1925.

observed by him it was inconceivable that this form had not been observed elsewhere. In 1930, in an article on Salzmann's nodular corneal dystrophy, one of the authors of this paper (D. K.[3]) reiterated Salzmann's opinion and stated:

> There can be no doubt that this type of corneal dystrophy is more common than only these two reports on the subject would lead one to believe, especially in view of the fact that Salzmann's group of cases were from Graz and its surroundings, and this case was seen in Chicago.

Since this statement was made this type of corneal disease has been described in one case by Ladekarl in Denmark and in nine cases by Lugli in Italy, and Salzmann wrote, in a personal communication in December 1933, that he had seen seventeen additional cases since his original publication. A total, therefore, of fifty-two cases of Salzmann's nodular corneal dystrophy have been recognized by Salzmann, Ladekarl, Lugli and ourselves.

In 1930, Ladekarl [4] of Copenhagen, in a report on two atypical cases of Groenouw's nodular corneal dystrophy, emphasized the clinical similarity of his second case to those described by Salzmann. Ladekarl's patient, a woman aged 30, had had mild symptoms of ocular irritation at irregular intervals from childhood to the time she was 24 years old. At this age she became aware of a bilateral reduction of her visual acuity. In the following six years the condition progressed, and when the patient was seen by Ladekarl there were four prominent, somewhat translucent, whitish-gray opacities in the lower half of the right cornea. Viewed with the slit lamp, these gave the impression of subepithelial nodular formations. Minute superficial vessels were seen to extend from the limbus to the nodules. The posterior half of the cornea was normal. In the left cornea the nodules were confined almost exclusively to the central zone and covered practically the entire pupillary area. The partial conglomeration of the nodules formed a vaulting protuberance above the surface of the cornea.

Lugli,[5] of Bologna, in 1930 reported three cases, and in a second report [6] in the following year his series of cases had increased to nine. These nine cases were observed by him between October 1926 and December 1930. Five of the patients were females and four were males.

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3. Katz, Dewey: Salzmann's Nodular Corneal Dystrophy: Report of a Case, Arch. Ophth. **4**:16 (July) 1930.

4. Ladekarl, P. Möller: Two Atypical Cases of Keratitis Nodosa Groenouw, with Histological Examination, Acta ophth. **8**:213, 1930.

5. Lugli, L.: Delle distrofie corneali ed in particolare della forma di Salzmann, Bologna, Licinio Cappelli, 1930.

6. Lugli, L.: Distrofia nodulare della cornea (forma di Salzmann), Boll. d'ocul. **10**:216, 1931.

Although there was no active manifestation of keratoconjunctivitis in the majority of these cases, a previous keratoconjunctivitis could be diagnosed from the history and the objective examination.

Salzmann, in 1925, noted that the frequency of this ocular disease in the patients at his clinic was 1 : 2,420. Lugli, in 1931, found that the frequency in the patients at the ophthalmic clinic of the University of Bologna was 1 : 1,761. Salzmann's nodular corneal dystrophy has been recognized twice in seven years in the ophthalmic clinic of the University of Chicago. The ratio is 1 : 7,020.

REPORT OF A CASE

History.—Mrs. E. B., aged 24, was first seen in the ophthalmic clinic of Billings Hospital, University of Chicago, on Oct. 30, 1933. The unaided vision in the right eye was 0.1. This was improved to 0.5 by a manifest refraction. The unaided vision in the left eye was 1.5 + 3.

The patient was healthy until she was 5 years of age, at which time she had an attack of whooping cough. Three weeks after this illness she was taken to a dispensary because the right eye teared. This was not associated with secretion, pain or redness. She was treated twice weekly for three weeks. One week after the last treatment, the lids became agglutinated. They were separated by the patient's mother, and the eye was irrigated with a solution of boric acid. The lids were agglutinated every morning for a week, and photophobia and tearing were present during the day. The patient secured tinted lenses for these symptoms, wore them for one month and then discontinued their use as the symptoms had disappeared. At no time since then has either eye been photophobic, red or painful. The visual acuity was first tested during the course of a routine examination at school when the patient was 13 years of age. At this examination, she was for the first time aware that the visual acuity of the right eye was much less than that of the left. She again went to an ophthalmic dispensary but was not informed as to the nature of the lesion which had produced the reduction of the visual acuity. A "white ointment" was put in the conjunctival sac, and the eye was massaged. The treatment was discontinued after two weeks, and the patient has not had an ocular examination or treatment since. She is of the opinion that the vision of the right eye is no worse than when it was tested eleven years ago, and she has not observed any progression of the corneal lesions, which are subsequently described, since she was first made aware of their presence.

The patient's mother and father are living and are not aware of any ocular disease. There is no history of consanguinity. There are three sisters, one older and two younger than the patient, and a younger brother, none of whom has any "eye trouble." The patient's only child, 3½ years of age, has normal eyes. The patient is not aware of any ocular disease in her aunts or uncles.

Objective Examination of the Eyes.—The lids, margins of the lids, cilia and tarsal, fornix and bulbar conjunctiva of each eye were normal. The ocular movements were normal, and coarse horizontal nystagmoid movements were present on extreme abduction. The right palpebral fissure measured 8 mm.; the left, 9 mm. The right cornea was of normal size and shape and, except for the elevation of the nodules, was of normal curvature. The pathologic changes were so located

that the cornea was divided for descriptive purposes into a peripheral, a middle and a central zone (fig. 1).

The peripheral zone, varying from 2 to 4 mm. in width in the different meridians, was diffusely hazy and contained superficially situated vascularized tissue. There were no discrete opacities in this zone, but at its central margin there were four elongated, grayish-white, somewhat soft-appearing but definitely elevated masses —dystrophic nodules.

These dystrophic nodules were in the middle zone. They formed a discontinuous, somewhat oval ring. The dystrophic nodule in the upper temporal quadrant was 4 mm. long; a second was 1.25 mm. long and was located in the 12:30 o'clock meridian; a third extended from 2 to 4 o'clock and was somewhat horseshoe-shaped with its convex surface directed toward the nasal limbus; a fourth was 2 mm. long, with its long axis at right angles to the limbus at 7 o'clock. The width of these nodules varied from 0.5 to 2.25 mm. The two larger nodules, the one in the upper temporal quadrant and the other in the nasal quadrant, had a somewhat segmented appearance, as though they might have been formed by the union

Fig. 1.—Photograph of the right eye, showing the size, shape and location of the dystrophic nodules.

of smaller nodules. Each nodule had a dense white central portion and a less dense peripheral portion. No definite structural plan or characteristically formed substance could be made out in the nodules. They were translucent when examined by indirect light, and by this means of examination there was visible a granular stippling in their central portion.

In the central zone there was an extremely fine nebulous superficial opacity. The underlying structures were readily seen through this area.

Slit-lamp examination of the right cornea gave the following results: The epithelium in the peripheral zone was intact and contained many well defined, irregularly distributed, vesicle-like changes in the surface layer. The vascularized opaque tissue in this zone lay immediately beneath the epithelial layer. It did not produce any visible elevation of level of this zone above that of the central zone. The central margin of the peripheral opacity terminated in a rather irregular but well defined border and, where the dystrophic nodules were present, the opacity was seen to extend to their medial sides. The blood vessels entered the cornea in all meridians, but there was a definite tendency for them to converge toward the nodules. Some of the vessels remained superficial in their course through the nodules, but the majority passed through the bases of the nodules and on into the central zone. There were a few deep blood vessels. These extended to the central zone, where frequently their lumens became obliterated and only the collapsed walls of the vessels were present.

The dystrophic nodules protruded from the surface of the cornea to a marked degree (fig. 2). Their walls did not come directly forward from the cornea but arched gracefully. The nodules extended into and through the anterior one third of the corneal stroma. The thickness of the cornea at the site of a dystrophic nodule was one and one-half times that of the normal. This thickening would therefore amount to about 0.5 mm. There was no definite regular structure visible in the dystrophic nodules, but the central portion was clearly of a denser white than the peripheral portion. There was no other area in the cornea which could be considered a potential focus for the development of a fifth nodule.

The central zone of the cornea was of normal thickness. There was a nebulous opacity in the superficial layers. Some of the blood vessels in this zone had collapsed walls.

The corneal nerves, where seen, were of normal thickness. No abnormal nerve structures were seen entering the dystrophic nodules. There was no evidence of intra-ocular inflammation, past or present.

Fig. 2.—A slit-lamp view of the lower end of the "horseshoe-shaped" nasal nodule.

Slit-lamp examination of the left cornea revealed the following picture: Superficial blood vessels entered the cornea in all meridians and extended centrally for a distance of 2 mm. except above. Here there was a diffuse vascularized opacity immediately beneath the epithelium, and it extended centrally from the corneoscleral junction for 4 mm. The blood vessels terminated a little beyond the horizontal, well defined central edge of this opacity. Isolated and structurally different was a well outlined superficial opacity, 1.5 mm. in diameter, located a little below and nasal to the apex of the cornea. This opacity as well as the vascularization were suggestive of a previous phlyctenular keratoconjunctivitis.

The iris of each eye was normal. The pupils were from 3 to 3.5 mm. in diameter, and the direct, consensual and convergence reactions were normal. The lenses and disks were normal. The right disk was seen as readily as the left. The tactile tension of each eye was normal. The right cornea was definitely hypesthetic over all the dystrophic nodules but showed normal sensitivity throughout the rest of its extent.

General Examination.—Physical examination gave negative results. The neurologic findings were all normal. The Wassermann and Kahn tests of the blood were negative. The white blood count, hemoglobin and urine were normal. There

was no local, focal or general reaction following the subcutaneous injection of 1 mg. of old tuberculin. Following the subcutaneous injection of 3 mg. of old tuberculin there developed at the site of the injection an area of redness 7 cm. in diameter in the center of which was an area of induration 3.5 cm. in its vertical diameter and 4 cm. in its horizontal diameter. The patient recorded her temperature at four hour intervals following each injection. The highest recorded temperature was 99.6 F. A roentgenogram of the chest showed no evidence of parenchymatous tuberculosis, past or present. There were no calcified hilus glands visible.

Operation.—On Dec. 5, 1933, with local anesthesia (p-n-butylaminobenzol dimethylamino-ethanol hydrochloride-pantocaine) the nodule in the upper temporal quadrant was easily excised with a small Graefe knife. The tissue was not compressed by forceps or other instruments at any time. The nodule was secured in one piece, and the dissection appeared to follow along a plane of cleavage which did not become more superficial or deeper as the dissection progressed. After removal, the dystrophic nodule showed no tendency to shrink but retained its shape and form. It was bisected at a right angle to its length, fixed and embedded in paraffin.

Postoperative Course.—The patient experienced some ocular pain the night of the operation, but none thereafter. There was no local reaction to the procedure, and when the cornea was examined two days postoperatively the shallow depression at the former site of the nodule was covered by epithelium and did not stain with fluorescein. A thoroughly satisfactory slit-lamp examination of this area was impossible as the floor of the shallow depression was hazy, but no corneal nerves were seen in the area.

On Jan. 7, 1934, with p-n-butylaminobenzol dimethylamino-ethanol hydrochloride anesthesia, the horseshoe-shaped dystrophic nodule in the nasal quadrant as well as the nodule above it was dissected from the cornea. The dissection did not extend into normal transparent corneal stroma. Some of the epithelium in the central zone of the cornea was removed, following which this faintly but diffusely hazy area became transparent. The patient experienced some ocular pain and discomfort for twenty-four hours.

Ointment of yellow mercuric oxide (2 per cent) and ethyl morphine hydrochloride (5 per cent) were used postoperatively. When examined five months later, the patient stated that the unaided visual acuity of the right eye was better than it had been, and a manifest refraction improved it to 0.8—1. At this time the site of the former temporal nodule was on the same plane with the surface of the surrounding cornea except the very uppermost and lowermost ends. These areas were slightly elevated. The former site of the horseshoe-shaped nodule was elevated throughout except in its central portion. The elevation was not as marked as that of the original nodule. The temporal nodule had been more completely removed than the nasal. In the six months since removal there has been no evidence of nodular formation at the site of the former temporal dystrophic nodule, whereas it was apparent at the end of three months that there was a recurrence of the pathologic process at the site of the nasal nodule. The central zone became somewhat more transparent.

Histologic Examination of the Excised Nodules.—The excised dystrophic nodules were fixed in various solutions, embedded in paraffin, sectioned and then stained by various methods.[7] The pathologic changes characteristic for one

7. Dr. P. A. Delaney, Douglas Smith Foundation for Medical Research, department of pathology, University of Chicago, aided in the preparation and study of the pathologic material.

nodule differed strikingly from the changes present in either of the other two nodules, as noted in the following histopathologic descriptions.

The nodule located in the 12 o'clock meridian was fixed in a dilute solution of formaldehyde, U. S. P. (1:10), and the whole tissue was stained with Ehrlich's acid hematoxylin and scarlet red. There was no microchemical evidence of fat.

Fig. 3.—Section of tissue from the nodule in the 12 o'clock meridian. Reduced from a magnification of 650.

The tissue was then embedded in paraffin, cut and stained with hematoxylin and eosin (fig. 3).

The epithelial layer throughout its greatest extent was three cell layers thick. The cells were flattened and showed pyknosis, and the cell walls were not visible. There were a few wandering cells. Immediately beneath the epithelial layer a homogeneous structure closely resembling Bowman's membrane was present for

short stretches in a few areas. The superficial lamellae (anterior third of the specimen, corresponding to about one-sixth the thickness of a normal cornea) were parallel to the surface and were straighter and narrower than normal corneal

Fig. 4.—Section of tissue from the nodule in the nasal quadrant; × 300.

lamellae, and a faint fibrillation was visible when they were examined under high magnification and with reduced light. There were few spaces between the lamellae in this area, and the whole area appeared fused and condensed into a

clear-staining, almost homogeneous mass. In the posterior two thirds of the specimen the lamellae coursed obliquely and were wider and much less compact, with many more spaces and a somewhat more definite fibrillation. The nuclei were very sparse in the anterior third of the specimen and, although more numerous in the posterior two thirds, even here they were much less frequent than in the normal corneal stroma. The nuclei were elongated, densely stained and filled with chromatin. The protoplasmic processes were long threadlike and also deeply stained. There were no wandering cells, blood vessels or foreign substances.

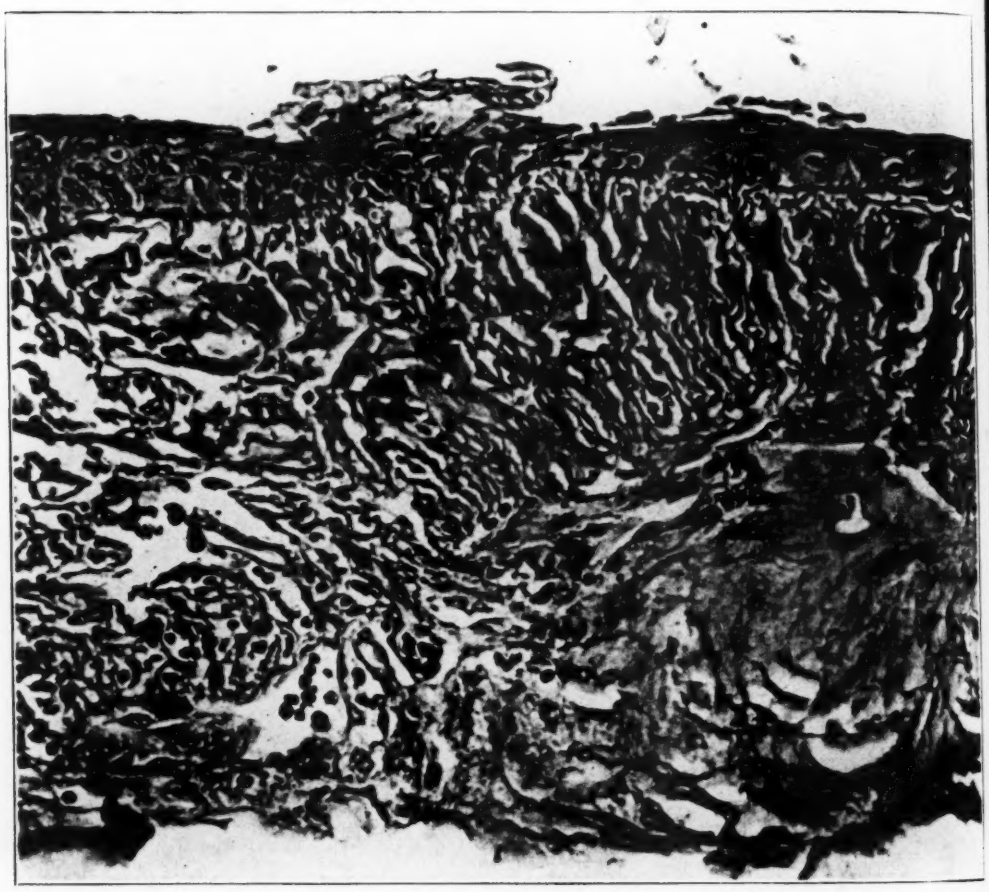

Fig. 5.—Section of tissue from the nodule in the upper temporal quadrant; × 325.

The nodule of the nasal quadrant was placed in Bouin's fixative [8] and stained by the method of Masson—iron hematoxylin, acid fuchsin-ponceau red and aniline blue (fig. 4). The epithelium varied in thickness from four to ten or more cell layers. The surface cells were flat. In some areas the outer two to

8. The composition of Bouin's fixative is as follows: 2 per cent trichloracetic acid, 1 part; 40 per cent solution of formaldehyde neutralized with magnesium carbonate, 5 parts, and saturated aqueous solution of trinitrophenol, 15 parts.

four layers consisted of flat cells. Where the epithelium was thinnest, there was marked pyknosis of the cell nuclei in all the layers present, and the cell outlines were indistinct. With increase in thickness the epithelial cells became more distinct, with variation in shape from the flat surface cell to the polygonal cell in the middle and deeper layers. In some areas the basal cells were smaller than those immediately above, and in other areas the basal cells were flat. Intercellular edema was more distinct and the intercellular bridges more conspicuous as one passed from the surface to the basal cell layer. An occasional wandering cell was present in the basal cell layer. Bowman's membrane was absent throughout. The epithelium was separated from the underlying stroma by extremely fine granular particles. In the superficial layers of the stroma there were areas in which the nuclei were prominent, and the related fibrous connective tissue was pale in contrast to other areas immediately adjacent, where the nuclei were less numerous and the connective tissue much more dense. The stroma was compact. The fibrillae had diverse directions with no tendency to lamellar formation. The cellular structure consisted of nuclei with marked degenerative changes. Some of the nuclei, still large and frequently linear, were pyknotic. The majority were swollen and vacuolated and had little chromatin. Very little extranuclear cytoplasm could be defined. Beneath this area there were some lamellae with a direction quite parallel to the surface. There were no wandering cells or blood vessels. There were no foreign elements present.

The dystrophic nodule of the upper temporal quadrant was fixed in acetic acid, osmic acid and potassium bichromate (fig. 5) in which fixative neutral fat, if present, stains black. The cut sections were stained with aniline acid fuchsin and methyl blue.

The epithelium varied in thickness from two to ten cell layers. The change in thickness was gradual. The cell borders were distinct. The cells of the superficial layer were flat and contained narrow, elongated nuclei. Some of these cells were cornified. The nuclei in the cells of the second layer were oval, with their long axes parallel to the surface. The nuclei in the deeper layers were somewhat rounded and frequently contained from two to four nucleoli. The cells of the basal layer were not columnar, as is the normal basal cell layer of the cornea, but were similar in form to those superficial to them. The nuclei in some of the cells in the outer layers were pyknotic. Intracellular edema, appearing as a halo about the nucleus, was present in many of the cells; others showed numerous vacuoles in their cytoplasm, and some were completely vacuolated. The intercellular bridges were plainly seen in areas. Only an occasional wandering cell was present. Bowman's membrane was absent throughout. The connective tissue beneath the epithelium was split up to a marked degree and formed a loose network with the majority of the component parts somewhat perpendicular to the surface. At one end of the section there were distinct thin-walled blood vessels with prominent lining endothelium. In this area there was a moderate diffuse cellular infiltration. Some of these cells appeared to be macrophages; others were round cells, and there were a few free red blood cells. Beneath this region of split-up lamellae there was a relatively thick area of dense fibrous connective tissue poor in nuclei in which the fibrillae had diverse directions with no tendency for lamellar formation. There were no fat-staining substances, nor were foreign substances such as calcium present.

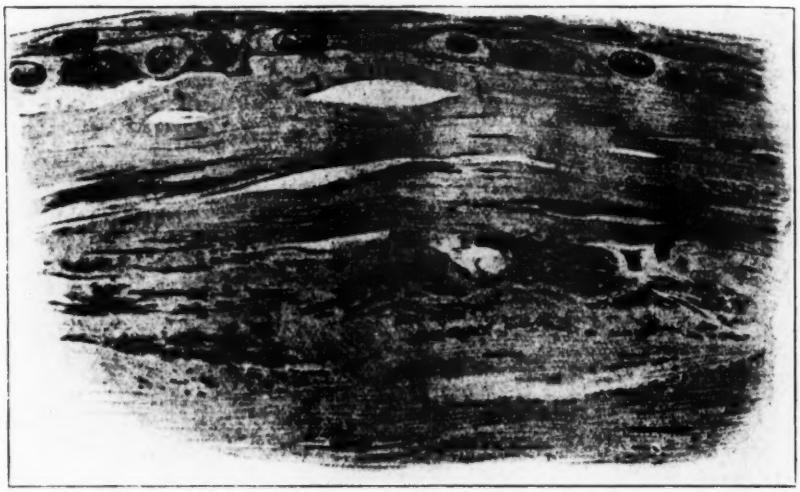

Fig. 6.—The histopathologic changes in the nodule removed by Salzmann.

Fig. 7.—The histopathologic changes in a portion of one of the nodules removed by Ladekarl.

REPORTS OF EXAMINATION OF NODULES BY OTHER AUTHORS

The only two other pathologic reports of nodules of Salzmann's nodular corneal dystrophy are by Salzmann and Ladekarl, and they differ markedly from each other.

Salzmann, in his histopathologic study (fig. 6), found that mechanical injury of the surface had produced a thinned epithelial layer. Bowman's membrane was absent. The superficial layers of the removed stroma were composed of old scar tissue. The fixed cells were few and irregularly distributed. In the deeper layers, irregular areas of corneal lamellae were heavily stained by hematoxylin and eosin. Lamellae which took the normal hematoxylin-eosin stain were present between the more heavily stained areas. The lamellae were swollen and wavy and were obliquely situated. Strongly stained granules were present in this layer as well as in the adjacent deeper layers. The deepest layers of the excised piece were composed of normal corneal stroma.

Ladekarl's sections (fig. 7) showed an epithelial layer which varied in thickness. There were areas with the normal number of cell layers and some areas with only one layer of flattened cells devoid of distinct nuclei. Basal columnar cells were not present. There were no distinct cell borders. The nuclei were poor in chromatin and were vesicular and vacuolated. Karyolysis had occurred here and there. The epithelium was frequently detached from the under layer in those areas where it was thinnest. Bowman's membrane was found to be intact in the few areas in which the epithelium and stroma appeared to be normal, but where the transitions to a thin epithelial layer were present it was frayed and disappeared in the network of the stroma. The changes in the stroma appeared restricted to the more superficial layers and were of two types, partly a splitting up of the lamellae giving rise to a network texture with long strands parallel to the surface and partly a transformation to a mass of a more homogeneous aspect, as though it were edematously swollen. In the split-up areas there was a slight increase of corneal cell nuclei. These were located for the most part along the sides of the connective tissue strands in the network and were frequently larger in size and lighter in shade than the normal, as though they were edematous. A few wandering cells were present in one area only. When stained by Van Gieson's method, the aforementioned homogeneous areas were slightly lighter red than normal corneal stroma and contained only a few isolated weakly stained thick nuclei. Lakekarl was of the opinion that these lighter homogeneous areas were becoming hyalinized though from the various staining preparations this could not be definitely determined.

The descriptions by Salzmann and Ladekarl have little or no pathologic similarity, and were it not for the fact that there were many clinical features common to both, justifiable doubt that these two cases were of the same clinical entity would exist. Certain gross pathologic similarities exist between the changes in the first nodule in the case reported here and that described by Salzmann, and between the third nodule and that described by Ladekarl. That Salzmann's nodular corneal dystrophy can be diagnosed only by its clinical history and appearance and not by its pathologic changes is definitely established in the pathologic study of the nodules removed in the case now reported.

COMMENT

The three nodules in the case reported here showed changes which were constant for each nodule but varied markedly from those in the other. In spite of this variation the pathologic descriptions of the three nodules have been arranged in what we believe to be the chronological order in regard to the development of the pathologic changes; thus, the first nodule shows the early changes which take place in this disease; the changes in the second nodule are later, and those in the third nodule are still later. In this respect, it is unfortunate that neither the patient nor her relatives are able to state when the dystrophic nodules first made their appearance and that they were much less able to state in what order they appeared.

In general, we believe that the small isolated nodule was the more recent and that those formed by the apparent coalescing of several nodules, as in the upper temporal quadrant and in the nasal quadrant in this case, were older. This is substantiated only by our interpretation as regards the sequence of the pathologic changes present in the nodules.

The study of the case shows rather conclusively that in the earliest stage the epithelium is thinned and consists of flat cells with pyknotic nuclei. In a later stage the epithelial layer varies in thickness and consists of flat and polygonal cells with cornification of some of the superficial cells, marked pyknosis and intracellular and intercellular edema in the other layers. In the latest stage the variation in the thickness of the epithelial layer is less pronounced, and the degree of cellular degeneration is more marked.

Bowman's membrane is destroyed by a gradual process of disintegration without inflammatory reaction or vascularization. Begun in the stage represented by the first nodule, it is completed in that represented by the second.

In the stroma the superficial layers are the ones first involved. The cells show evidence of pyknosis, and the intercellular connective tissue is condensed into a clear-staining, almost homogeneous mass. Later the stroma shows lamellar fibrillation and more marked cellular changes —pyknosis and vacuolation. No wandering cells or blood vessels are present. As the deeper layers become involved, the superficial layers show more marked degenerative changes with some cellular infiltration and vascularization. There was no deposition of foreign substances in any of the stages studied.

It can be readily seen that any number of gradations can, and no doubt do, occur between the normal state and the stage represented by the first nodule, between the first and the second nodule and between the second and the third nodule, and that many stages are possible beyond the one here represented in the third nodule.

In this case the marked differences in the pathologic changes in the nodules removed from the same cornea at about the same time (interval of one month) are therefore significant and furnish a probable solution to a number of questions which have been raised in regard to the pathologic process of nodular corneal dystrophy. Slit-lamp examination of the nodules revealed essentially the same biomicroscopic picture and gave no suggestion that the nodules differed so markedly histopathologically.

It is, of course, probable that the histopathologic changes in this condition do not progress with equal rapidity in the various nodules, nor are the parts involved—epithelium, Bowman's membrane and stroma—affected to the same degree in all the nodules of approximately the same stage. Thus one is likely to note marked discrepancy in the pathologic findings in various cases of this clinical entity. If this is correct it is not surprising that the pathologic reports of this corneal nodular dystrophy by Salzmann and Ladekarl have little resemblance to each other. It is quite probable that this is also the reason why no two pathologic reports on nodules removed in Groenouw's nodular corneal dystrophy are alike in spite of the relatively numerous pathologic reports on this condition.

E. Fuch's statement in reference to the nodule which he saw develop in certain cases of eczematous keratitis is most interesting. He thought that they might be due to a keloid formation of the underlying scar. Kaufmann,[9] the general pathologist, stated:

> The completed, typical keloid is presented as a homogeneous mass of closely approximated, broad bundles of fibers, between which spindle-shaped cellular elements are present. The fibers may be arranged parallel to the surface and to the long axis of the tumor.

A hypertrophic scar shows essentially the same pathologic change. The microscopic changes present in the first nodule in the case reported simulate those of a hypertrophic scar or completely developed keloid. Each of these processes invariably develops on the site of a previous injury involving the dermis. If in Salzmann's nodular corneal dystrophy the early pathologic process is a localized hypertrophic one which has developed on the site of a previous pathologic change due to phlyctenular keratitis, it would readily account for the prominence of the nodules, for with the hypertrophy there would be a bulging forward of the anterior stroma and the development of the clinical picture typified by Salzmann's nodular corneal dystrophy, in which the outstanding feature is the elevation of the nodule.

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9. Kaufmann, E.: Pathology for Students and Practitioners, translated by Stanley P. Reimann, Philadelphia, P. Blakiston's Son & Co., 1929, vol. 3, p. 2175.

TREATMENT

Therapeutically it is well known that medicinal treatment alone—usually ethyl morphine hydrochloride and ointment of yellow mercuric oxide—is of no avail in nodular corneal dystrophy either of the Salzmann or of the Groenouw variety. A spontaneous cure has never been reported. Some authors have recorded favorable results following the excision of the nodules in cases of Groenouw's dystrophy. Other authors have reported a rapid reproduction of the nodules following excision, some becoming more extensive and more opaque. In Ladekarl's case of Salzmann's nodular corneal dystrophy the condition responded to surgical removal of the nodules with an improvement of the visual acuity. From Salzmann's experience it is known that cauterization with a Paquelin cautery is likely to result in an extensive thick opacification of the cornea with pronounced loss of vision. In the case now reported the combination of surgical and medical treatment has produced an improvement of vision, and the patient states that the visual acuity is continuing to improve. It is not known why nodular corneal dystrophy is beneficially influenced by surgical treatment in some instances and made worse in others. Is it not possible that the difference in the results may be ascribed to the completeness or incompleteness of the excision of the nodules? In the former instance the local seat of the primary changes is removed, whether it is the result of a nutritive disturbance or a disease of the terminal nerve plates, whereas in the latter instance not only does the focus remain but it is probable that sometimes the local influence is irritated or stimulated by the incompleteness of the surgical procedure so that the nodules rapidly reproduce themselves and even become more extensive. As stated previously, following cauterization in one of his cases Salzmann was painfully surprised to see that the opacification of the cornea rapidly became more extensive and denser. In view of the fact that the nodules extend into the anterior stroma, it does not appear likely that they can be completely removed with the cautery.

A suggested method which is most likely to remove the nodule or nodules completely without perforation of the cornea is as follows: With a small Graefe knife, that is, one which has been sharpened several times so that it is about one-half its original size, all of the nodule above the surface of the cornea as well as part of the portion which extends into the corneal stroma can be removed readily and safely. To remove the base completely, the use of a trephine, which is not too sharp, together with a Bowman knife, should give good results. The trephine is used to cut through the various layers of the pathologically involved corneal stroma. These can then be removed with the Bowman knife and an Elliot scleral disk forceps. The thickness of the removed tissue will

depend on the depth at which the cornea is no longer opaque. The trephining is repeated in whatever direction is necessary until the floor of the dystrophic nodule becomes completely transparent.

SUMMARY

This is the fifth report of Salzmann's nodular corneal dystrophy. First described by Salzmann in Austria, it has since been reported in the United States, Denmark and Italy, this being conclusive evidence that special local conditions are not the cause of the development of the disease. The reports have all substantiated Salzmann's observations as well as his belief that this corneal dystrophy is not a rarity or confined to any special part of the world. Nothing of a positive nature regarding the fundamental etiologic factor has been definitely established. All the theories—hereditary transmission, nutritive disturbance, trigeminal degeneration, etc.—advanced to explain the etiology of Groenouw's nodular corneal dystrophy might be considered plausible explanations for Salzmann's nodular corneal dystrophy, but the findings to date do not permit a positive statement regarding any one fundamental etiologic factor, except that invariably the corneas affected show changes of previous attacks of phlyctenular keratitis.

The histopathologic changes observed have varied strikingly in the three pathologic studies thus far reported. It is doubtful if any two specimens of this condition from two different nodules will ever be secured which will be identical in their histopathologic changes.

CONCLUSIONS

The pathologic process of Salzmann's nodular corneal dystrophy is a noninflammatory, slowly progressive process, early hypertrophic and later degenerative, producing marked and varied alterations of the epithelial layer, Bowman's membrane and the outer one third to one half of the corneal stroma. All descriptions of the histopathologic changes present will necessarily be a description of a stage of the disease.

Salzmann's nodular corneal dystrophy should be treated surgically. The indications are a reduction or threatened reduction of visual acuity. At present complete removal of the nodule or nodules holds out the only hope for successful treatment.

LIPIN INTERSTITIAL KERATITIS

PARKER HEATH, M.D.
DETROIT

The condition under consideration may be defined as a deep interstitial fat and cellular infiltration of the cornea (usually vascular) and part of a disturbance in fat metabolism. The process is usually protracted, painless and bilateral, though a long interval may separate involvement of the second eye.

EARLY, ACTIVE LESION

The deep central area of the cornea or its margin may be affected first. Careful biomicroscopic methods usually show a deep peripheral feed in the central lesions. The earliest changes consist of fine droplets, varying in size, and cells infiltrating the substantia propria, associated with loss of transparency anterior to Descemet's membrane, and associated with endothelial pitting. A deep vascular loop composed of an entering small artery and a larger returning vein pushes in, preceded by an arc of loss of transparency. Adjacent to the loop may be seen the minute droplets. The cell stream in the early and active lesions is rapid and fairly uniform. The lesion expands with more vascular penetration and branching and infiltration. There is an increasing loss of transparency like a nebulous gray cloud outside of the areas of denser stromal storage. A few dots of pigment are seen on the endothelium and lens capsule. At the borders of some infiltrations are seen minute areas, faintly and irregularly yellowish. A small lesion may reach maturity, regress and disappear. The early process may extend over many weeks or months.

MATURE LESION

With further advance of the disease process the entering vessel loops remain deep at the periphery and more anterior toward the anterior pole. With a greater infiltration a posterior stromal swelling and endothelial cell wall form. Optical section of the cornea may show a posterior bulging and a reflection ring. The entire process is characteristically posterior, though with older lesions the anterior third of the cornea may be involved. It is also characteristic that the epithelium is not disturbed unless the activating agent is anterior, as herpes. The aqueous beam shows occasional cell clumps, particularly with high power arc illumination. The corneal nerves seem fewer and larger than usual.

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Fig. 1.—*A*, deep ingrowing vascular loops of the active lesion (retro-illumination); small artery and larger vein accompanied by minute droplets, preceding cloudy loss of transparency and exudate (semidiagrammatic). *B*, nearly mature lesion (optical section) showing different levels of infiltration and posterior swelling of the cornea (semidiagrammatic).

The conjunctival vessels, particularly in the older patients, show at the limbus exaggerated irregularity in caliber and course. One of these large, fairly straight, bright conjunctival vessels dips posteriorly at the outer limits of the limbus. Numerous deep, straight, parallel, light pink-colored vessels are the principal invaders. The severity of the process probably varies with the number of vessels, and recovery is more prompt and complete with fewer vessels. Older stored lipins show more yellowish coloration and a mat, like that seen magnified in rag paper. Cell types may be studied from material withdrawn by a needle. Tissue studies have shown small lymphocytes, occasional polymorphonuclears, extracellular fat crystals and large pale histiocytes storing fat. The anterior stroma near the limbus usually remains clear. High power magnification shows the infiltration to be flocculent and partly amorphous; within a mat of crystals—a crystal felt—a few doubly refractile bodies are seen. A large involvement of the cornea has a few anterior tortuous vascular twigs and heavy posterior congestion. The light lemon color of lipochromes is irregularly disposed. A faint, broken, brown ring may sometimes be seen, somewhat like the Kayser-Fleischer ring, but not so regular or so peripheral. Fresh material withdrawn by aspiration shows much fat-storing substance with crystals and fat-laden histiocytes. Tissue studies (Bachstez,[1] Jaensch,[2] Kolen,[3] Heath[4]) are well known and show the characteristic cells and storage material, although in the past different interpretations have been put on these findings. Frequently there is diminution in corneal sensibility. A variable mild, relatively aplastic uveitis is characteristic, apparently mostly anterior. A few small clumps of pigment may be scattered on the anterior lens capsule. The reactions of the iris to light are diminished, and mydriasis is less prompt and complete than normal. An extensive involvement of the cornea is associated with a proportionate increase in uveitis.

RETROGRADE LESION

The retrograde lesion shows a diminishing storage and posterior cell wall and an interrupted segmental cell stream within the vessels. Storage material leaves the anterior layers irregularly, and masses may be seen lingering with vascular twigs. The interlamellar substance is more crystallized and crystal-bearing and contains fewer histiocytes and more plasma cells. Characteristic of this stage, when there has been extensive involvement, are folds of Descemet's membrane, both straight and crossing, and the branching relinquished paths of blood vessels. Also

1. Bachstez: Wien. med. Wchnschr. **71**:1044, 1921; Ztschr. f. Augenh. **54**:36, 1924.

2. Jaensch: Fat Degeneration of the Cornea, Arch. f. Ophth. **119**:198, 1927.

3. Kolen: Virchows Arch. f. path. Anat. **263**:46, 1927.

4. Heath: Ocular Lipid Histiocytosis and Allied Storage Phenomena, Arch. Ophth. **10**:342 (Sept.) 1933.

a large lesion in the retrograde stages shows anterior concentric rings not unlike a series of senile arcs one within the other. The outer ring is often partly in the position of the usual senile arc. Useful vision may result in the child and younger adult from a cornea previously opaquely infiltrated. A variable irregularity of the posterior corneal surface is noted in the healed stage. Also fine, thin, fibrous scarring and stripes in the deep substantia propria and some yellow pigment remain. Scarring in some areas has a honeycomb appearance and shows Descemet's warts. Characteristic old paths of blood vessels, loops and remnants are usually present. In some corneas the late lesions have anterior vascular overgrowths and epithelial and subepithelial dot-like and fine linear opacities. These patients have less visual recovery.

Fig. 2.—Nearly mature lesion, optical section, shows different levels of infiltration and posterior swelling of the cornea (semidiagrammatic).

ETIOLOGY

The causes producing these lesions lie in the field of liporeticular disease. In both the misnamed "primary corneal dystrophies" and those lipin changes secondary to trauma or infection, as herpes, the underlying pathologic process is the same. In general there is an overwhelming lipid cellular response to a mild activating lesion. In the so-called primary types there probably exists a constitutional faulty metabolism of fat, and a mild unrecognized uveitis is part of the pathologic process. The vascular arrangements, lowered temperature, exposure to repeated mild trauma and laminated structure peculiar to the corneal tissue all contribute to its delinquency. The aging process, an excessive intake of fat, a disturbed water balance, deficiencies in carbohydrate metabolism,

endocrine disturbances, circulating poisons, and toxins from local and distant sources of infection are all etiologic possibilities. Any mechanism precipitating or coarsening the normally fine fat emulsion can produce a foreign-body type of reaction with great cellular response. It is the immune overresponses by cells and storage which make the lesion so obstructive to sight. The material comes from without and is not a dystrophy of corneal tissue. The smaller infiltrations are rather common. The largely involved thickened cornea is rare. The interstitial infiltration is more rapid and diffuse in onset in the child, and slower and more a fixed storage in the older adult.

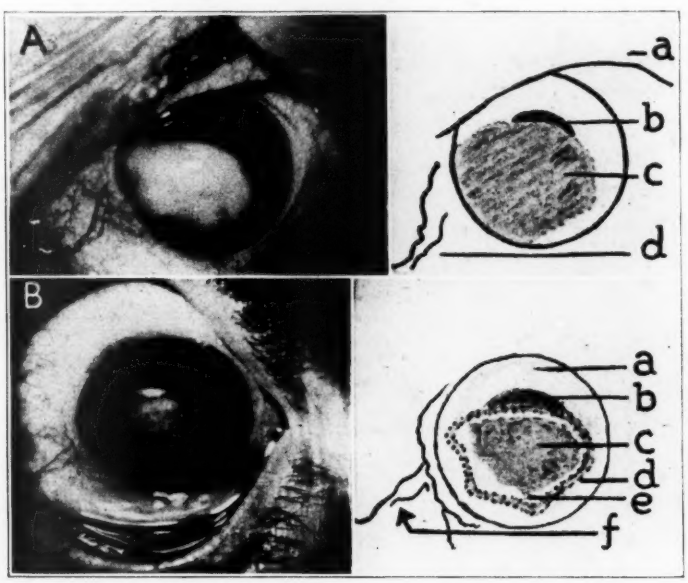

Fig. 3 (case 1).—*A*, active lesion in left eye at six months: (*a*) lid, (*b*) pupil, (*c*) infiltrate, (*d*) conjunctival vessels. *B*, retrograde lesion in left eye at ten months: (*a*) senile arc, (*b*) pupil, (*c*) infiltrate, (*d*) outer ring, (*e*) clear interval, (*f*) conjunctival vessels.

TREATMENT

No doubt some lipin interstitial corneal infiltrations are mild and short lived, and spontaneously disappear. Cases of transient (hourly changes) loss of corneal transparency, without vascular ingrowth, have been reported (Aubineau [5]). Noothoven van Goor and Schaly [6] stated that Espagnet (1888) reported such a case. The joint authors describe

5. Aubineau, E.: Ann. d'ocul., **159**:580, 1922.

6. Noothoven van Goor, J. M., and Schaly, G. A.: Nederl. tijdschr. v. geneesk. **75**:1422 (March 21) 1931.

a syndrome found in cases with hypophyseal tumor, namely, xanthoma diabeticorum generalisatum, corneal edema with hypercholesteremia and diabetes and neurologic ileus. The combination of high blood sugar and fat soon leads to disastrous consequences. The other extreme is seen in massive infiltrations lasting years in the active state. Treatment should be directed toward removing possible activating agents. A search should be made for foci of infection, disturbances in metabolism, particularly in that of fats and carbohydrates, and endocrine imbalances. A low intake of fat and carbohydrate is insisted on. The patient's environment is changed as a means of increasing his bodily activity. Daily walking as an exercise with a substantial minimum has been adopted. Biweekly sweats and massage, thyroid by mouth and insulin are of value. A chart kept by the patient, cataloging intake and output, is of value. Local heat by Shahan thermophore, electric pads and diathermy have been used. Protection against minor trauma is obtained by the

Fig. 4 (case 2).—Old healed lesion: (*a*) pupil, (*b*) scar, (*c*) pupil.

use of glasses and removal of concretions on the lids. The eyes have been kept under the influences of atropine and scopolamine. Early treatment is most successful. In the severe cases useful vision is not lost unless treatment is begun so late that the corneal cellular deposits have been converted into a dense fibroblastic scar. If the process runs a mild course and regresses spontaneously in two or three months, it is likely that active treatment will hasten recovery and give an improved end-result.

DIFFERENTIAL DIAGNOSIS

It is my opinion that a group of corneal lesions now separately classified as separate entities may be grouped more accurately and understandingly as lipin interstitial keratitis. Among these [7] are: some forms of parenchymatous keratitis; keratitis centralis annularis; keratitis profunda and the misnamed fat "dystrophy" of the cornea. "Xanthomatosis

7. Vogt, A.: Lehrbuch und Atlas der Spaltlampen Mikroskopie des lebenden Auges, Berlin, Julius Springer, 1930, vol. 2.

of the cornea" describes a stage of fat keratitis. Included is the large group in which the process is activated by trauma, retained foreign body, herpes and infection, provided these agents merely initiate the serious pathologic storage process. Related are arcus senilis and senile marginal atrophy. With understanding study of the cornea biomicroscopically the pathologic picture largely classifies itself. Lipidous or xanthomatous bulbi show typical golden flakes in the cornea and lipocrystalline deposits in the filtration angle, as well as substantial inflammatory changes in the iris, sclera, etc., and cholesterol crystals can be aspirated from the anterior chamber in large numbers. Syphilitic interstitial keratitis, while deep, has more numerous groups of vessels; the exudate is patchy; it has more scattered pigment and, of course, has all the inflammatory types of cells and associated changes. Purely senile changes in Descemet's membrane and the endothelium are usually avascular and patchy. Keratitis disciformis probably represents an avascular form of lipin keratitis, and the vascular ingrowth is absent, meager or late. Many of the so-called crystalline degenerations probably represent a late scar stage of lipin infiltrations with a particular combination of those substances. The appearance of the lesion varies with the proportions and kinds of fat substances present. Some forms of hypopyon lead to stromal opacity from the disturbances of the endothelium that are set up. Studies of blood cholesterol have not been satisfactory. Few of the cases reported have shown excessively high fasting blood cholesterol. Studies of blood cholesterol represent this substance only, and the perhaps more important fats are not estimated. A less selective estimation of blood lipin would be more valuable.

SOME GENERAL IMPRESSIONS

As indicated, the younger the patient, all else being equal, the more rapid and complete the recovery. A retrograde lesion may become active by a large intake of fat. Females are more susceptible than males. A mild uveitis is present intermittently. Severe advancing monolateral inflammations, unless treated early, become bilateral. Mild forms frequently spontaneously recover. Studies of blood cholesterol give incomplete information regarding blood fats. Most patients with active lesions have excess weight, lessened physical activity or both. No marked changes in intra-ocular tension have been observed. No typical lesions of the fundus or field changes and no racial tendencies have been noted. In elderly patients an association with xanthomatous skin lesions has been noted.

TUBERCULOUS AND STREPTOCOCCIC RETINAL HEMORRHAGES

CHARLES M. SWAB, M.D.
OMAHA

As the cases that gave rise to the question underlying this discussion do not conform in detail to a given classification, the title may seem to be rather broad, so far at least as the use of the epithets "tuberculous" and "streptococcic" apparently implies or assumes the cause. That in the instances to be cited a definite relation exists between retinal hemorrhages on the one hand and tuberculous and streptococcic infection on the other, the evidence seems to show, and it is the purpose of this communication to set forth such evidence. I realize fully and appreciate the position of those who maintain that in a specific case it is far from easy to show beyond reasonable doubt that the streptococcus was the cause of the retinal hemorrhage. As a justifiable hypothesis, however, and merely for the sake of argument, one may premise that streptococcic focal infections exist in certain persons suffering from ocular disorders; in the event that those foci are removed and recovery follows in due time, it is at least tenable that a definite cause and effect relationship may exist. That there are valid objections to this conclusion even when the clinical experiment has been successful, no one will deny.

Moreover, there has been a tendency on the part of physicians generally, especially in the past, to question the clinical diagnosis of intra-ocular tuberculosis. It required no little conviction and no small courage on the part of an ophthalmologist to maintain that an intra-ocular lesion was tuberculous in the face of the evidence of negative reports from the internist and from the roentgen and clinical laboratories. Seemingly, there was no appeal from the dictum of Collins [1] who insisted that the approved methods of establishing with certainty the diagnosis of intra-ocular tuberculosis are histologic observation of tuberculous structure in the material in question, or inoculation of experimental animals with resultant evidence of tuberculosis, or bacteriologic demonstration of the tubercle bacillus.

To follow Collins' [1] proposed routine in clinical cases is, under the circumstances, out of the question. In the vast majority of cases the

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1. Collins: Tr. Internat. Cong. Med., 1906, sect. 11, p. 62.

ophthalmologist is consulted by patients who are seeking relief from their present symptoms, and about the last thing in their minds at the time is the thought of a detailed or exact diagnosis. Thus, when a patient has an intra-ocular lesion suspected of being tuberculous and inaccessible for studies requiring laboratory finesse, the ophthalmologist must depend on other methods both for diagnosis and for therapy. This fact has made him a leader in the diagnostic and therapeutic uses of tuberculin. Research, clinical data and histologic observations have greatly enriched the knowledge of tuberculous perivasculitis with retinal hemorrhage, but the most far-reaching contribution to the subject has been made through the empirical use of tuberculin.

REPORT OF CASES

Five cases have been chosen for this presentation of the symptom complex of retinal hemorrhage occurring with tuberculosis and focal infection; they elaborate the points which I wish to emphasize.

CASE 1.—On Jan. 14, 1931, Mr. C. B., aged 30, a civil engineer, complained of blurred vision of the left eye. He had first noticed the blurring six days before. The family history was irrelevant. He had always been well; he worked out of doors most of the time, and he never had had any ocular trouble. There was no history of constipation, epistaxis or hemophilia.

The ocular examination showed that vision in the right eye was 6/9+, and in the left, 6/9+; each near point was 11 cm. The external ocular structures were normal. The fundus of the right eye showed a kite-shaped opacity in the superior region of the vitreous; the details of the fundus were normal. The fundus of the left eye showed a massive hemorrhage in the vitreous, which was for the most part dustlike in character; details of the fundus could not be seen distinctly.

The patient was hospitalized, and a medical survey was made. All the laboratory examinations gave normal results, and the physical findings were negative except for an apical abscess of one tooth and chronic tonsillitis. A Mantoux test with old tuberculin was positive, and therapeutic intramuscular injections were given. On January 27 there was a focal hemorrhage in the retina of the left eye resulting from the administration of 0.1 mg. of old tuberculin. As the tuberculin therapy had to be discontinued temporarily, the infected tooth was removed on January 30.

After five weeks the patient was discharged from the hospital. It was noted then that the vitreous had cleared somewhat, thus allowing a more satisfactory inspection of the fundus of the left eye; numerous hemorrhagic areas could be seen in the inferior and temporal parts of the retina. Soft, white perivascular exudates were observed along portions of the veins. It was now seen for the first time that there was an active process in the retina of the right eye; the pathologic change was restricted to the nasal periphery. In addition to hemorrhagic areas there was an area of proliferation; this lesion was slaty gray. The veins in that locality exhibited perivasculitis. By April 1, 1931, the dose of old tuberculin had been brought up to 0.4 mg., but at that time focal reactions occurred in both eyes. Fortunately the hemorrhages were small and were confined to the retinas. On April 20 tonsillectomy was performed. When the treatments with

old tuberculin were resumed the dose was reduced, and the patient again showed improvement. On October 23 the vision in the right eye was 6/6—1; in the left, 6/6—3. The old tuberculin therapy was not discontinued until March 11, 1932; six weeks later both eyes were healed, and the patient was able to return to work.

On April 13, 1934, the patient returned because of blurred vision in the left eye. Corrected vision in the right eye was 6/5; in the left eye, 6/6—4, blurred. There were no evidences of recent trouble in the right eye, but the proliferation in the nasal part of the retina was more marked than at the last examination. The lesion now showed definite elevation; some of the peripheral blood vessels ran into the lesion and were not seen again for a considerable distance, if at all. Coarse fibrous bands passed horizontally nasalward in the retina; in the left eye there was an extensive hemorrhagic area in the vitreous. No evidence of hemorrhage was visible in the retina. Because of the previous experience with this patient, old tuberculin was again given in small doses five days apart. By adhering to this regimen the patient was able to resume his occupation on May 27 with the hemorrhage in the vitreous practically all absorbed.

Fig. 1 (case 1).—Right eye, showing proliferative retinitis.

Case 2.—On Aug. 25, 1929, Sister Mary D., a teacher, aged 33, reported that vision in the left eye was reduced. The reduction of sight had occurred five weeks before and had come on suddenly. The family history was unimportant. The patient had been treated for pulmonary tuberculosis in 1925; otherwise her personal history was not relevant. There was no history of constipation, epistaxis or hemophilia. Corrected vision in the right eye was 6/9; in the left eye it was reduced to ability to count fingers at 0.66 meter. Except for a pallor of the conjunctivae, the external ocular structures were normal. An examination of the fundi showed that in the right eye the media were clear and the disk, vessels and retina healthy; in the left eye the media were clear and the disk was normal; in the macular region there was a rounded, elevated grayish mass about 2 disk diameters in size; in the nasal periphery of the retina there were several patches of heavy, slaty-gray exudate. Perivasculitis was noted in places in the nasal part of the retina. In the periphery below the disk there was an extensive area of retinitis proliferans or a fixed mass in the vitreous body; the mass extended upward into the vitreous so that vessels on its surface could be seen plainly with a plus 6 lens in the ophthalmoscope. Transillumination was dull but positive.

The patient moved to a different locality and was not seen again until October 2. At that time uncorrected vision in the left eye was 6/15. The fundus of the left eye showed one large opacity in the vitreous with strands adherent to it; the

macular area was flattened; just below the disk were two rounded white lesions which were thought to be choroidal tubercles or exudates in the outer layers of the retina. On account of the pulmonary condition no treatment with tuberculin was given. On June 20, 1930, corrected vision in the left eye was 6/9+.

On Sept. 12, 1932, there was a massive hemorrhagic area in the vitreous of the left eye; as before, vision was almost completely gone. The patient was sent to the hospital, and a general physical examination was made. A roentgenogram of the chest showed considerable peribronchial thickening in both pulmonary fields, accompanied by calcification of the lymph nodes; no pathologic changes were demonstrable in the parenchyma. Treatments with old tuberculin were then begun, and the vision improved steadily. On Feb. 3, 1933, corrected vision in the left eye was 6/9; the same vision was recorded on June 26, 1934.

CASE 3.—Dr. K. F., an intern, aged 25, was seen on Oct. 19, 1931; he complained of blurred vision in the right eye. This symptom had been present for almost a month. The family history was essentially irrelevant. There was no history of hemophilia, epistaxis or constipation. Vision in the right eye was

Fig. 2 (case 2).—Left eye, exhibiting satisfactory healing in the region of the disk and macula.

6/6—1, blurred; in the left eye, 6/6+. The external ocular structures were normal. An examination of the fundus of the right eye showed many dustlike opacities in the vitreous; the disk was slightly blurred above, but there was no swelling; the macular region was clear; the retinal veins were markedly tortuous and uneven in caliber; in places there was perivasculitis; in the superior temporal region of the retina there was an extensive hemorrhage. In places the veins appeared markedly foreshortened. The fundus of the left eye showed a few opacities in the vitreous; the veins were tortuous and uneven in caliber; there was no evidence of hemorrhage. After being hospitalized for seven days the patient broke out with measles. As soon as his condition permitted, a nonvital tooth was extracted; cultures were taken from the area of the root and Streptococcus viridans was grown.

The Mantoux test and therapeutic tests with old tuberculin were negative. An autogenous vaccine which had been prepared was administered. On December 5 vision in each eye was 6/6+. The periphlebitis was markedly improved; a few perivascular streaks remained, and there were occasional dilatations in the veins. The retinal hemorrhage was almost completely absorbed, but it had left its mark; a 3 by 5 degree absolute scotoma could be outlined 15 degrees to the left and downward from fixation. Improvement continued, and there was no recurrence. On

June 23, 1934, both eyes were normal except for an area of retinochoroidal degeneration and atrophy in the superior temporal region of the fundus of the right eye.

CASE 4.—Miss M. B., a nurse, aged 22, was first seen on Aug. 12, 1931. The day before she had noted blurred vision in the left eye. The family history was not pertinent, and there had been no previous ocular trouble; the patient had had catarrhal jaundice two months previously; there was no epistaxis, hemophilia or constipation. Vision in the right eye was 6/9 +4; in the left, 6/9 —3. The external ocular structures were normal. The fundus of the right eye was normal throughout; that of the left eye showed a few coarse floaters and many fine opacities in the vitreous and a small hemorrhagic area at the inferior nasal border of the disk; the retinal vessels appeared healthy. On September 1, new hemorrhagic areas were present at the temporal edge of the disk and below the fovea. The patient was hospitalized, and a medical survey was made. Two days later there was evidence of a hemorrhage along the course of the inferior temporal vein, and there was more hemorrhage in the vitreous. The general physical examination, urinalysis, Wassermann test and blood count failed to reveal any significant data. Dental roentgenograms showed a suspicious appearance in one tooth. Intra-

Fig. 3 (case 4).—Left eye, showing a membranous sheath in the posterior vitreous directly in front of the disk.

dermal and therapeutic tests with old tuberculin were negative. On September 11 the questionable tooth was removed under aseptic precautions, and cultures were taken. Streptococcus viridans was grown in pure culture, and a vaccine was prepared.

Following the dental operation there was an increase in the small hemorrhagic areas in the retina; four new ones appeared along the course of the superior nasal vein. There was now an exudate in the retina just below the disk, and perivascular exudates were noted along the inferior temporal vein. After three weeks of rest in bed supplemented by treatments with the vaccine there was some improvement. By November 20 all of the blood in the retina had been absorbed. The patient was examined on June 25, 1934; at that time there was a dense fibrous sheath in the posterior portion of the vitreous which extended from the superior temporal border of the disk, down and in. This sheath was slightly above the level of the fundus and was stationary. Since it was directly in front of the papilla vision was not interfered with; in fact, vision in each eye without correction was 6/6.

CASE 5.—Mrs. E. H., an office clerk, aged 46, was seen on Feb. 7, 1933; she complained of blurred vision in the left eye. There had been no evidence of any

ocular trouble until about three days previous to this visit. The family history was irrelevant. The patient had had pleurisy at the age of 26. Tonsillectomy and total hysterectomy and salpingectomy had been performed in 1931. In 1932, the patient was injured in an automobile accident and was unconscious for three days. In this accident she suffered a fracture of the outer table of the skull in the left occipital region. No constipation, hemophilia or epistaxis had been noted. Vision in the right eye was 6/9 +4; in the left, 6/9. The external ocular structures were normal. An examination of the fundus of the right eye disclosed several fine weblike opacities in the vitreous; other details were normal except

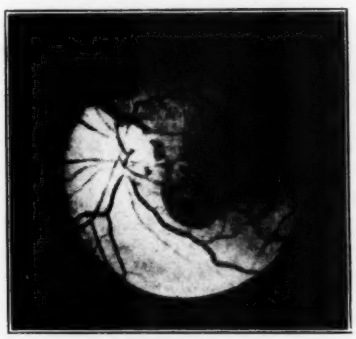

Fig. 4 (case 5).—Left eye, showing large preretinal hemorrhage which is supplied from two sources.

Fig. 5 (case 5).—The source of the blood above the disk is arterial; three branches of the superior temporal artery are completely occluded.

for a considerable degree of arteriovenous compression. The fundus of the left eye showed: numerous dotlike opacities in the vitreous; filmy hemorrhagic areas along the superior and temporal borders of the disk; some bleeding from the vessels just above the disk; a fairly large preretinal pool of blood, down and out from the disk; several completely occluded vessels—arterial terminals—in the superior temporal portion of the retina, and some oozing from the retina, up and out from the macula. The patient was sent to the hospital, where a general physical examination was made.

The blood pressure was 160 systolic and 110 diastolic; the blood count was normal; the urinalysis showed a mild degree of pyelitis; the Wassermann reaction of the blood was negative; a roentgenogram of the chest demonstrated no patho-

logic changes. Several teeth had a suspicious appearance; the tonsillar stumps revealed infection, and there was an infected mass of adenoid tissue in the nasopharynx. In spite of a positive Mantoux test, I was misled as to the etiology. On February 23 nine teeth were extracted, and some necrotic bone was removed. Cultures were made from the three oral areas that were attacked; Streptococcus haemolyticus was grown from each focus. Five days later the tonsillar stumps and adenoids were removed. The patient left the hospital on March 8. The blood pressure at that time was 135 systolic and 95 diastolic; the general condition seemed good; the ocular condition remained as it had been. Treatment with an

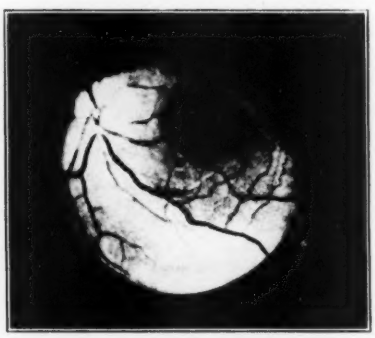

Fig. 6 (case 5).—Left eye after improvement. The hemorrhage continues, but the reservoir is smaller. Note the droplets of blood just above the crescent.

Fig. 7 (case 5).—Left eye, showing proliferations in the retina.

autogenous vaccine failed to aid the retinal condition. With the advent of more evidence of hemorrhages in the retina injections of tuberculin were begun on April 3. It was possible to control the amount of blood in the large preretinal pool, but as fast as any was absorbed new blood ran in; the preretinal reservoir was fed by the hemorrhages above the disk and by those which were situated above the macula. The lesion at this time resembled an oriole's nest (fig. 4).

By July 25 the dosage of old tuberculin had been increased to 6 mg. every fourth or fifth day. Some oozing still persisted for months; the preretinal hemorrhage was not all absorbed until Jan. 6, 1934. The proliferative changes in the superior and superior temporal parts of the retina were unusual. In one place, three white globular masses stood out; these fibrosed balls were in close proximity

to other proliferative changes. These were red clusters, which on close scrutiny appeared to be twists or loops of capillaries that projected above the level of the fundus.

The patient resumed her work on Feb. 1, 1934, but has been unable to discontinue treatment with tuberculin. On two different attempts to reduce the dosage too rapidly, new hemorrhages occurred in the superior part of the retina. At present, when 0.8 mg. of old tuberculin is given every fourth or fifth day the bleeding is controlled. A reduction of the quantity of tuberculin or a lengthening of the interval between treatments causes new hemorrhages. The use of bovine tuberculin is being considered.

COMMENT

Of the five cases reported in this article not one included a history of constipation, hemophilia or epistaxis. Primary vascular disease was not a factor except possibly in case 5. When this patient was first examined the retinal vessels showed some evidence of hypertension, and there was thrombosis (with obliteration of the lumen) of some of the branches of the superior temporal artery. On the other hand, the active pathologic changes in this patient were centered in the superior temporal quadrant, and the arteries were the source of most of the hemorrhages. The same observation was made by Derby,[2] and by Kyrieleis [3] in cases of recurrent retinal hemorrhage of tuberculous origin. The ages of the five patients reported on in this article range from 22 to 46; three were women, and two were men. In three patients the left eye alone was affected; in one, the left eye only was at first affected but later the right eye showed hemorrhagic areas in the retina associated with an extensive retinitis proliferans; in one patient the pathologic changes were limited to the right eye except for a mild phlebitis in the retina of the left eye.

Cases 1, 2 and 5 have been classified as tuberculous, since therapy with old tuberculin brought about improvement while other treatment was of no avail. In cases 3 and 4 the condition cleared up after the removal of streptococcic focal infections and treatment with autogenous vaccines. Two of the patients with tuberculous lesions have been treated for a recurrence of retinal hemorrhage, while one has had intermittent bleeding in the retina for over a year; the others have had no recurrence. In one of the cases of focal infection (case 3) the vitreous became entirely clear; in the other (case 4) there has occurred as a late change a dense, gray fibrous sheath in the posterior part of the vitreous body. The three patients with tuberculous lesions all exhibited some form of permanent organization in the vitreous, while the retina showed some degree of proliferation. The latter manifestation did not occur in the other patients. In all these patients the retinal vessels were involved; in the four younger persons mainly the veins were affected, while the

2. Derby: Tr. Am. Ophth. Soc. **19**:238, 1921.
3. Kyrieleis, W.: Arch. f. Augenh. **107**:182, 1933.

older patient appeared to have had more arterial than venous involvement. None had any detectable pathologic changes in the anterior segment of the eyeball.

In reviewing the published accounts of cases which present the symptom complex of hemorrhage into the retina and vitreous one finds that various theories have been offered as to the etiology. Eales [4] ascribed the cause in his cases to a neurosis which exhibited itself through the sympathetic nervous system, affecting the vessels of the alimentary canal by a vasomotor contraction and those of the retina by dilation. Zentmayer [5] suggested the possibility that the endocrines, particularly the suprarenal glands, are factors in this condition. A somewhat similar suggestion was made by Wilmer [6] in 1921 in a discussion of the paper of the late Dr. Finnoff.[7] Young [8] reported cases of recurrent hemorrhage into the retina and vitreous which he attributed to a deficiency in calcium. Parsons [9] is a supporter of the theory of deficiency in calcium.

Although the symptom complex of recurring hemorrhage into the retina and vitreous was described by von Graefe [10] eighty years ago, it was not until Noll's [11] article was published in 1908 that a tuberculous etiology was suggested. Since that time, however, many writers have presented cases in which tuberculosis was considered to be the chief factor in the production of the hemorrhages. Among the contributors were Axenfeld and Stock,[12] de Schweinitz and Holloway,[13] Knapp,[14] Jackson,[15] Finnoff,[16] Jackson and Finnoff,[17] Holloway [18] and many others. In our day it may be said that the advocates of the tuberculous origin of such cases greatly outnumber those who favor any other etiologic factor. As Jackson [19] has pertinently stated, however, there is probably more than one cause for this symptom complex. A considerable number

4. Eales, H.: Birmingham M. Rev. **3**:262 (July) 1880; Ophth. Rev. **1**:41, 1882.
5. Zentmayer, W.: Am. J. Ophth. **3**:652, 1920.
6. Wilmer: Tr. Am. Ophth. Soc. **19**:238, 1921.
7. Finnoff: Tr. Am. Ophth. Soc. **19**:238, 1921.
8. Young: Tr. Am. Acad. Ophth. & Oto-Laryng., 1929, p. 191.
9. Parsons, J. H.: Diseases of the Eye, ed. 6, New York, The Macmillan Company, 1931.
10. von Graefe, A.: Arch. f. Ophth. (Abt. 2) **1**:294, 1855.
11. Noll: Arch. f. Augenh. **63**:213, 1908.
12. Axenfeld and Stock: Klin. Monatsbl. f. Augenh. **49**:28, 1911.
13. de Schweinitz and Holloway: Ophth. Rec. **19**:243, 1910.
14. Knapp: Arch. Ophth. **42**:1, 1913.
15. Jackson: Ophth. Rec. **25**:1, 1916.
16. Finnoff: Ann. Ophth. **26**:329, 1917.
17. Jackson and Finnoff: Am. J. Ophth. **2**:715, 1919.
18. Holloway, T. B.: J. Iowa M. Soc. **15**:629, 1925.
19. Jackson: Tr. Am. Ophth. Soc. **19**:238, 1921.

of cases presumed to be due to focal infections have been reported by Ellett,[20] Radcliffe and Young,[21] Redding,[22] Lewis,[23] Finnoff [7] and Godwin.[24] In these cases foci were located in the teeth, tonsils and sinuses; in one case Finnoff [7] considered the gastro-intestinal tract as a focus.

CONCLUSIONS

Tuberculous and streptococcic retinal hemorrhages are essentially similar in onset and in the character of the active lesions in the retina and vitreous. The diagnosis can be established by clinical experiment; in some cases the condition clears up on treatment with tuberculin; in others, after removal of focal infections and treatment with autogenous vaccine. From the cases which have been reported, together with those here presented, it seems that retinitis proliferans is a common sequel in cases of tuberculous origin. The proliferation varies with each case. In the two cases reported in this article, in which the etiologic factors were streptococcic focal infections, the retina did not exhibit proliferative scar tissue.

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20. Ellett: Am. J. Ophth. **6**:423, 1923.
21. Radcliffe and Young: Am. J. Ophth. **6**:496, 1923.
22. Redding, L. G.: Atlantic M. J. **27**:640, 1924.
23. Lewis, F. P.: A Bacterial Toxin as the Cause of Retinal Hemorrhage, J. A. M. A. **70**:1813 (June 15) 1918.
24. Godwin: Am. J. Ophth. **10**:171, 1927.

GLIOMA OF THE OPTIC NERVE

REPORT OF A CASE

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ROSARIO, ARGENTINE REPUBLIC

Glioma of the optic nerve is the blastoma originating in the glial or neuroglial tissue of the optic nerve, and it has nothing to do with the tumor which used to be called glioma of the retina, a clinically and pathologically malignant neoplasm which develops during the first years of life.

One must insist on this distinction because for years confusion has existed concerning the classification of these tumors, and only by repeatedly emphasizing the difference between the two neoplasms can the old idea be destroyed.

In very early embryologic stages the nervous system is represented by a channel arising from the ectoderm, and gradually the walls of this channel approach one another until a tube, the medullary groove, is formed. The walls are formed by cellular groups called neurospongia, and the inside is lined with an epithelial sheet, the neuro-epithelium. The neural tube widens in its distal portion, thus constituting the cerebral vesicle—anterior, middle and posterior. From two opposite points of the anterior vesicle develop two new vesicles, the optic vesicles. Each of these vesicles is lined with the continuous neuro-epithelium. Finally the epithelium of the optic vesicle develops into the pigmented sheet and the ten layers of the retina.

Originating from the neuro-epithelium there are cellular elements which are the potential functional centers of the nervous system, and deriving from the neurospongia there are elements of protection and support, such as: the meningoblasts, which will form the meninges, the Schwann cells and the sheaths of the same name, which envelop the nerve fibers, and the neuroglia cells with their fine and multiple ramifications, which constitute the neuroglia, the tissue which serves as a frame and support to the ganglionic cells and to the nerve fibers.

That is why when speaking of glioma one must refer to the blastoma derived from the neuroglia, a tissue formed by astrocytes, oligodendrocytes and microgliacytes derived from the neurospongia.

The so-called glioma of the retina is a tumor originating in the retinal cells, and it is known that those cells originate from the neuro-epithelium. This ontogenetic difference is not merely theoretical; the histopathology and the clinical evolution reveal much greater differ-

ences between the two. Glioma of the optic nerve is generally benign, but glioma of the retina has a malignant evolution.

All this does not imply that the glial elements which serve as a support to the retinal cells may not produce intra-ocular tumors with the characteristics of glioma, that is, with differentiated cells and fibers of neuroglia. Ascunce has reported a case of such a tumor, properly classified under the heading of glioma, but the tumor which occurs in children and which is clinically called glioma of the retina is of neuro-epithelial origin, and though the name glioma of the retina is still used to designate it, this tumor differs from the true glioma in origin, evolution and malignant character.

Having established that difference clearly, I shall report a case of glioma of the optic nerve.

REPORT OF CASE

J. A., a girl aged 19, was seen at the clinic on May 27, 1933. The family history was irrelevant; the past history showed that the patient had scarlet fever

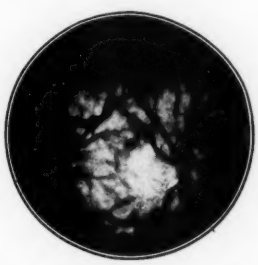

Fig. 1.—Fundus of the left eye.

at the age of 3 years and a gastro-intestinal upset accompanied by fever at the age of 9. Following this it was noticed that the left eye protruded slightly. On examination the left eye protruded and was displaced downward and outward; readings by means of the Hertel exophthalmometer were 12 for the right and 24 for the left eye. There was no chemosis, no edema of the lids and no pulsation. The bony margins of the orbit were normal, but in the upper inner angle of the orbit there was a cystlike mass. The exophthalmos was not altered by pressure on the jugular veins, by exertion or by changes in posture. Roentgenograms taken in Rhese's position showed the left optic foramen to be twice as large as the right; its outline was distinct. The cyst was punctured and 4 cc. of bloody fluid removed; this procedure reduced the exophthalmos for twenty-four hours.

With the patient under rectal ether anesthesia, a Krönlein operation was performed by Dr. Cames. Above the optic nerve a cyst containing about 3 cc. of fluid was found, and below the cyst there was a tumor involving the whole optic nerve. The portion of the optic nerve between the globe and the optic foramen, including the tumor, was removed. Healing was uneventful. On July 6, 1933, the left eye was level with the right eye, and there was no exophthalmos. The disk was white and cupped, and the circulation in the fundus was as shown in figure 1. One year later word was received from the patient that no further change had occurred in the left eye.

COMMENT

The case reported does not constitute a problem of diagnosis. The exophthalmos was axial, direct, irreducible and slowly progressive, the eye maintaining power for all movements and the optic foramen being enlarged. All the characteristics of a benign tumor of the optic nerve were present. The main interest lies in some facts revealed through the operation; these facts will now be commented on. In addition to the esthetic result obtained (fig. 2), which is important to a girl, the results of the operation prove two facts: (1) that although all the posterior ciliary arteries and the central artery of the retina were sectioned at the posterior pole the eye was still well nourished and (2) that although the ciliary nerves, which are considered to be the trophic nerves of the eye, were sectioned no destructive process took place.

Fig. 2.—*A*, appearance of the eye before operation; *B*, appearance of the eye twenty days after operation.

Another fact which arrests attention is the persistence of the arterial circulation, which should cease following surgical section of the principal vessels. One must agree that as the tumor was growing it compressed the arteries until the collateral circulation was sufficiently well established to become the principal source of blood supply; thus the rôles became reversed, the collateral circulation serving as the chief source of blood for the eye.

The collateral circulation is carried on with the aid of the anterior ciliary arteries, branches of the muscular arteries, usually seven, which pierce the sclera 2 or 3 mm. behind the cornea and join the choroidal circulation, communicating with the retinal arteries through Haller's ring.

In the photograph of the fundus (fig. 1) taken thirty days after the operation one can see a rich vascular net formed by new vessels, and it is not easy to detect the situation of the normal vessels of the fundus.

In regard to the question whether the ciliary nerves are the trophic nerves of the eye, the fact that a section of the ciliary nerves did not interfere with perfect conservation of the eye has demonstrated the fallacy of that supposition.

In several cases in which vision was lost through hypertensive processes or other causes, I have injected alcohol in the area of the ciliary ganglion in order to relieve the pain and have obtained a functional result equivalent to section of the sensory fibers, but I have never noted the production of dystrophic alterations.

All ophthalmologists know the results of experimental section of the sensory nerves of the eye. Surgical section, as in the case reported here, has shown that it is an error to ascribe a trophic rôle to the ciliary nerves.

As a conclusion of practical interest one must keep in mind that the destructive lesions generally observed after section of the trigeminal nerve or after extirpation of Gasser's ganglion are probably due to traumatism or infection; these can easily be avoided with proper postoperative care.

SUMMARY

The etiologic and histopathologic difference between so-called glioma of the retina (malignant) and true glioma of the optic nerve (benign) is insisted on. A case of glioma of the optic nerve extirpated by Krönlein's operation is reported. One and a half years after operation the patient's eye was in excellent condition in spite of the sectioning of the central vessels and the ciliary nerves.

This fact demonstrates once more that the ciliary nerves are not trophic nerves. The collateral circulation has replaced that of the principal vessels.

Clinical Notes

DISLOCATION OF SOEMMERING'S RING

Report of a Case

M. W. Jacoby, M.D., and B. J. Wolpaw, M.D., Cleveland

In a comprehensive review of the American and foreign literature reports of only six cases of dislocation of Soemmering's ring can be found. These were reports by Tooke,[1] Schneider,[2] Wessely,[3] Poos [4] and Jess.[5] It is the rarity of the condition which prompts us to report our case.

In view of the recent thorough review of the condition by Tooke [1] we shall not repeat more than to state that Soemmering's ring is the ring of cortex contained between the anterior and the posterior capsule which remains posterior to the iris at the time of extracapsular extraction. Following the operation this ring may become detached from the zonular ligament and may be dislocated either anteriorly or posteriorly. The condition occurs most frequently in myopic eyes.

REPORT OF CASE

J. D., a white man 66 years of age, had a cataract in the left eye in 1904. A combined extracapsular extraction was performed at the Budapest Ophthalmologic Clinic in 1905. Following the operation the patient considered his vision good until 1918, when a white ring appeared in the anterior chamber of his eye. From that time on vision gradually failed until only light was perceived. No history of trauma could be obtained.

Fourteen days prior to admission the eye became red and painful. Examination revealed marked conjunctival and ciliary injection. There were numerous new vessels in the cornea. The corneal epithelium was edematous. There were no deposits on the posterior surface of the cornea. The anterior chamber was very deep, and there was a coloboma of the iris at 12 o'clock. In the anterior chamber was a white ring, broken only over the coloboma. The ring floated freely around the entire circumference except at the coloboma. A dense capsule membrane was present in the pupillary space. It was impossible to see the fundus. Tension was

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From the Ophthalmologic Service of St. Luke's Hospital.

1. Tooke, F.: Dislocation of the Ring of Soemmering: Its Removal, Brit. J. Ophth. **17**:466 (Aug.) 1933.

2. Schneider, C. O.: Soemmering Ring Luxated into the Anterior Chamber, Am. J. Ophth. **10**:273, 1927.

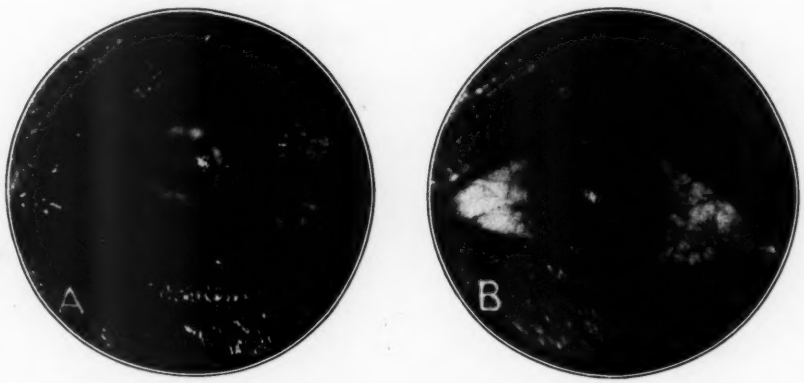
3. Wessely, K.: Arch. f. Augenh. **66**:277, 1910.

4. Poos, F.: Klin. Monatsbl. f. Augenh. **86**:449 (April) 1931.

5. Jess, A.: Klin. Monatsbl. f. Augenh. **86**:98, 1931.

22 mm. of mercury (Schiötz). With warm compresses and mild astringents the reaction subsided; however, the eye at the end of four weeks' observation was still painful and red. It was thought that the reaction would continue as long as the ring was in the anterior chamber, and surgical intervention was advised.

Through a 6 mm. keratome incision at the superior limbus the ring was withdrawn except for a small fragment which broke loose and receded into the vitreous. A moderate amount of fluid vitreous escaped. There was little postoperative reaction. The wound healed rapidly. Four weeks after the operation it was possible to see faint details of the fundus. The eye was myopic.



A shows the ring in the anterior chamber prior to operation, and *B*, the eye after the ring was removed.

Eight weeks after operation a large ulcer developed which involved two thirds of the cornea. This was resistant to treatment for several weeks, slowly healing with a resulting dense corneal scar.

The eye at present is painless and without any sign of irritation. Vision is limited to perception of light.

THE CALCULATION OF LOSS OF VISION

J. A. C. GABRIELS, M.D., ALBANY, N. Y.

In the report of the Committee on Definition of Blindness,[1] the value of vision is expressed in terms of the fractional or decimal equivalent of the Snellen symbol, e. g., 20/200 or 6/60 or 0.1. This is at variance with the visual ratings as proposed by the Committee on Compensation Tables for Eye Injuries—a committee of the Section of Ophthalmology of the American Medical Association. Whether the Snellen formula represents a true fraction has long been a source of controversy. I have repeated elsewhere [2] proof of the inherent fractional nature of the

1. Report of the Committee on Definition of Blindness, J. A. M. A. **103**:1445 (Nov. 10) 1934.

2. Gabriels, J. A. C.: The Problem of the Snellen Symbol, J. A. M. A. **101**: 872 (Sept. 9) 1933.

Snellen notation. Such recognition, as pointed out, does not warrant its acceptance for measuring the value of vision. Adoption of values based on the unit of surface rather than on the subtending visual angle or linear unit is again suggested as offering the only solution to this problem.

Briefly, what one determines with the Snellen test is the ability of an eye to distinguish a surface detail subtending equal dimensions—in other words, the surface of a square. For the norm this dimension is the angle 5 minutes of arc, or its component 1 minute of arc. The Snellen method employs for this purpose a letter or similar character having such dimensions. To conform with the requirement of the Snellen test, a standard test object is so constructed that the 5 minute as well as the constituent 1 minute angle, or minimum separable, implies a surface area corresponding, the length and breadth of which are equal, and withal inseparable. Hence the unit of surface rather than the coincident linear unit becomes the determinant in measuring the value of vision. This principle is in no way altered by the use of other test characters, such as, for instance, the broken ring, square, etc. The Snellen letter or numeral affords adequate opportunity for measuring the resolving power of an eye, and, it may be added, as a test of visual acuity it is scientifically sound and, when accurately interpreted, as physiologically true as any test; it has, moreover, the added advantage of universal appeal.

The actual method of calculation, citing a few of the notations in common use, is as follows:

Sn. 20/30 or 6/9 is 2/3, that is, a loss of 1/3—1/3 squared (1/3 × 1/3) equals 1/9 equals 0.111 equals 11.1 per cent visual loss or a visual value remaining of 88.9 per cent.

Sn. 20/40 or 6/12 is 1/2, that is, a loss of 1/2—1/2 squared (1/2 × 1/2) equals 1/4 equals 0.25 equals 25 per cent visual loss, or a visual value of 75 per cent.

Sn. 20/100 or 6/30 is 1/5, that is, a loss of 4/5—4/5 squared (4/5 × 4/5) equals 16/25 equals 0.64 equals 64 per cent visual loss, or a visual value of 36 per cent.

Sn. 20/200 or 6/60 is 1/10, that is, a loss of 9/10—9/10 squared (9/10 × 9/10) equals 81/100 equals 81 per cent visual loss, or a residual visual value of 19 per cent.

If one proceeds in this same manner, any visual symbol, whatever the designation, may be easily and quickly evaluated. Systematic study of such formulas and their values indicates clearly the character of the curve arising from losses affecting central or macular vision.

530 Madison Avenue.

DEVICE FOR TRAINING OF CHILDREN TO OVERCOME STRABISMUS

R. Townley Paton, M.D., New York

It has been my experience that many of the devices for the training of squinting eyes are too complicated for children of preschool age or that they fail to hold the child's attention for a sufficient time to make

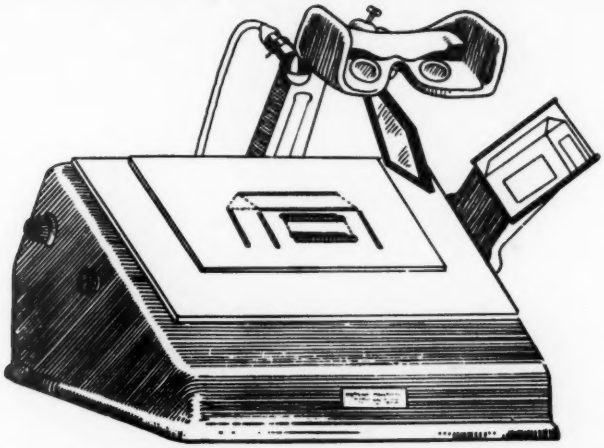


Fig. 1.—Maddox cheiroscope with card and stencil in position.

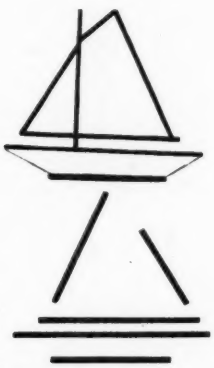


Fig. 2.—Examples of cards and stencils.

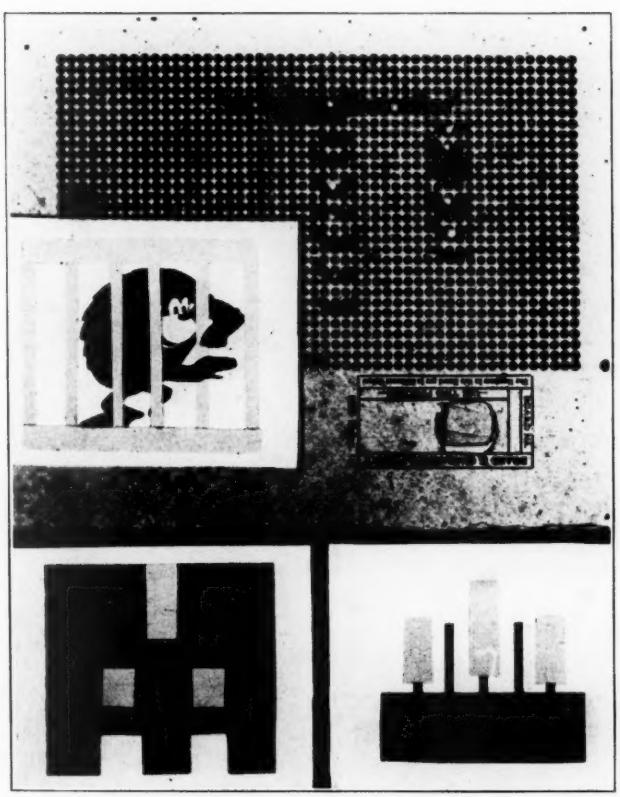


Fig. 3.—The punch board and sample cards.

the exercises worth while. The importance of developing the coordination of movements of the hands and eyes has long been recognized in the training of these patients. Exercises to stimulate correct orientation and projection are of fundamental importance, but most of the exercises are based on the supposition that the child can handle a pencil. A large percentage of children are unable to acquire the ability to hold a pencil for tracing exercises before the age of 4 or 5 years. Unless the child has acquired a considerable degree of dexterity, tracing exercises become tedious. Much valuable time may be lost in waiting for the child to learn to manipulate a pencil.

Therefore, an inexpensive series of cards [1] was devised to be used on the Maddox cheiroscope. These cards are made up in sets of two. One card of each set shows the picture drawn in a series of straight lines. The other is a stencil reproduction with some of the minor details left out. This is placed on the drawing platform (fig. 1). With a number of small sticks cut in lengths corresponding to the slots in the stencil, the patient is instructed to build the picture which he sees (fig. 2).

Another similar series was devised in which a punch board is placed on the drawing platform. For this exercise the child uses small pegs in place of the sticks and inserts them along the lines which he sees on looking through the objective (fig. 3). In order to avoid confusion, a piece of white paper may be set over the board and the pegs easily pushed through it.

It is surprising to see how quickly a child will perform these exercises. Once the projection has been developed, the other stereoscopic exercises become easier. A variety of small figures and colors have been added to some of the cards to stimulate fixation and to arouse the patient's interest.

1. The cards may be obtained from A. Haustetter, Inc., 2 East Forty-Fifth Street, New York.

Ophthalmologic Review

OCULAR DIVERGENCE: ITS PHYSIOLOGY AND PATHOLOGY

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NEW YORK

I. PHYSIOLOGY

PHYLOGENETIC DEVELOPMENT

In studying any function of the human organism much can be learned from its appearance and development in the phylum, and one is justified in expecting that the rule will hold good in dealing with the function of divergence. As a function, however, it has been discussed little or not at all in the literature, and so one will have to assume the time of its appearance and postulate its subsequent development by a consideration of some allied function. Is there a function the existence of which predicates the existence of divergence and to the smooth working of which divergence is indispensable? The function of convergence seems to fill these requirements. Certainly, without divergence of some sort convergence would be a crippling liability rather than a biologic asset, and nature has arranged her progress to suppress rather than to accentuate biologic weaknesses. One may be fairly safe, then, in linking the faculty of convergence to its less well known antagonist and in assuming that the appearance of the two functions on the phyletic scale is simultaneous.

The elaborate structure of motor function was built on the humble foundation of "tropism," motion to or from a light. This dyscritic function remains postural in all animals below the amphibia.[1] As the eyes came forward from the side to the front of the head and the snout receded,[2] the visual fields overlapped and binocular vision appeared. The parallel ontogenetic stage begins in the human embryo of 7 mm. when the optic axes are in a straight line. At 9 mm. the angle is 160

From the Department of Ophthalmology, Columbia University College of Physicians and Surgeons.

1. Parsons, J. H.: Introduction to the Theory of Perception, London, Cambridge University Press, 1927.

2. Wood-Jones, F.: Arboreal Man, London, Edwin Arnold & Co., 1916. Smith, Elliot: Evolution of Man, New York, Oxford University Press, 1927. Collins, Treacher: Tr. Ophth. Soc. U. Kingdom **41**:10, 1921. Smith, Mott, Whitnall, Keith et al.: Tr. Ophth. Soc. U. Kingdom **45**:53, 1925.

degrees; at 16 mm., 120 degrees, and at 40 mm., 72 degrees.[3] Neurologically, this stage is marked by an increase in the proportion of the nondecussating fibers in the optic tract.[4] At first (a stage exemplified in rodents and ungulates) there are no conjugate movements and only a few nondecussating fibers. The illustration in figure 1 of stages at the top and bottom of the scale renders it unnecessary to recount the various steps.

It is important at this point to distinguish binocular vision, stereoscopic vision and binocular plus stereoscopic vision. For instance, when (before the close of the Eocene period) the eyes of a tarsioid finally

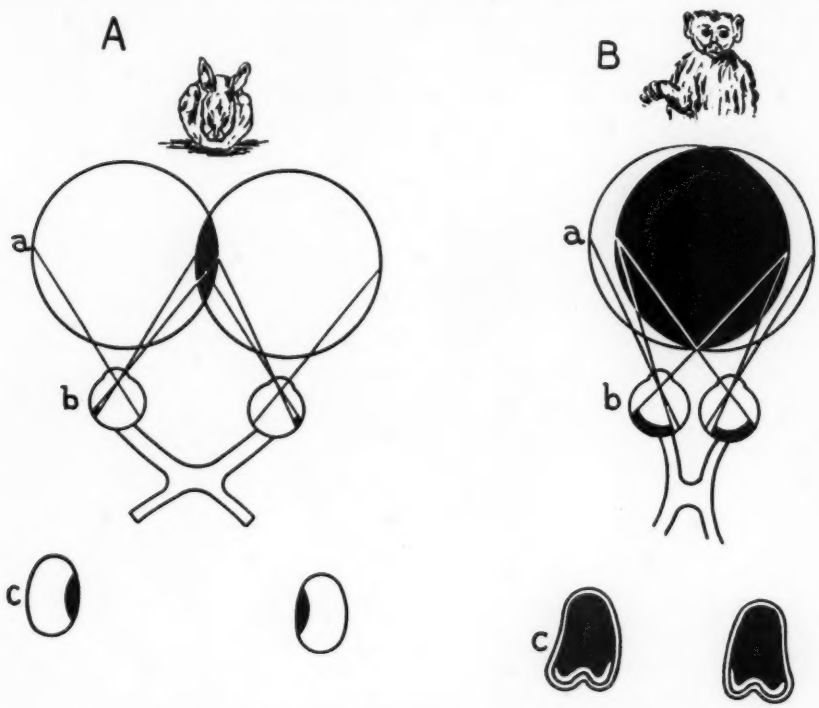

Fig. 1.—Diagrams showing the relation between the monocular and binocular fields of vision and their projection in the external geniculate bodies (*A*) in the rabbit and (*B*) in the monkey. *A* indicates the visual field; *B*, the retina, and *C*, the external geniculate body. The white area indicates the monocular field; the black area, the binocular field. (After Brouwer and Zeeman, Brain **49**:1, 1926.)

came around to the front, it acquired binocular vision, but no depth perception; and its descendant of today, Tarsius spectrum, is still under the necessity of moving the head through large circles in order to judge depth (fig. 2). Similarly birds of prey (in which binocular vision

3. Mann, Ida: Development of the Human Eye, London, Cambridge University Press, 1928.

4. Tilney, F.: The Brain from Ape to Man, New York, Paul B. Hoeber, Inc., 1928.

Fig. 2.—The spectral tarsier (after Poulton, in Smith's "Evolution of Man" [6]).

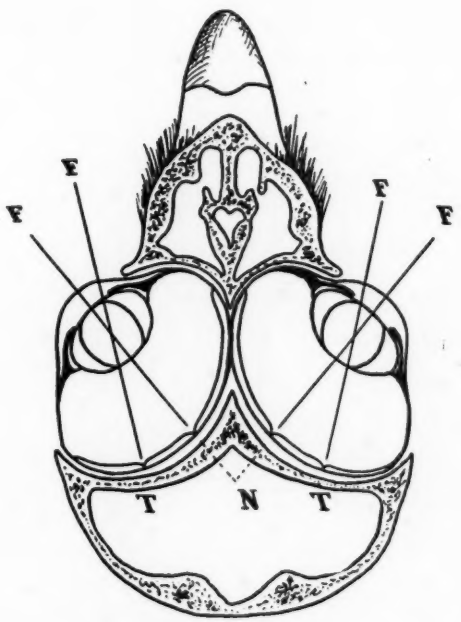

Fig. 3.—A bimacular eye, the eye of the broadwinged hawk (after Slonaker, in Wood, Casey: The Fundus Oculi of Birds, Chicago, Lakeside Press, 1917, p. 15). *F* indicates the fovea; *T*, the temporal region, and *N*, the nasal region.

is usually absent) have a well developed stereoscopic sense by virtue of two foveae in each eye (fig. 3). When one speaks of stereoscopic vision one should assume that the individual described as possessing that attribute has binocular plus stereoscopic vision, in that he has two central foveae of corresponding points and partial decussation of optic nerve fibers. This precocious development of the fovea was probably a differentiation of panoramic vision and was thus well advanced before being called on for new duties as the eyes came forward.[5]

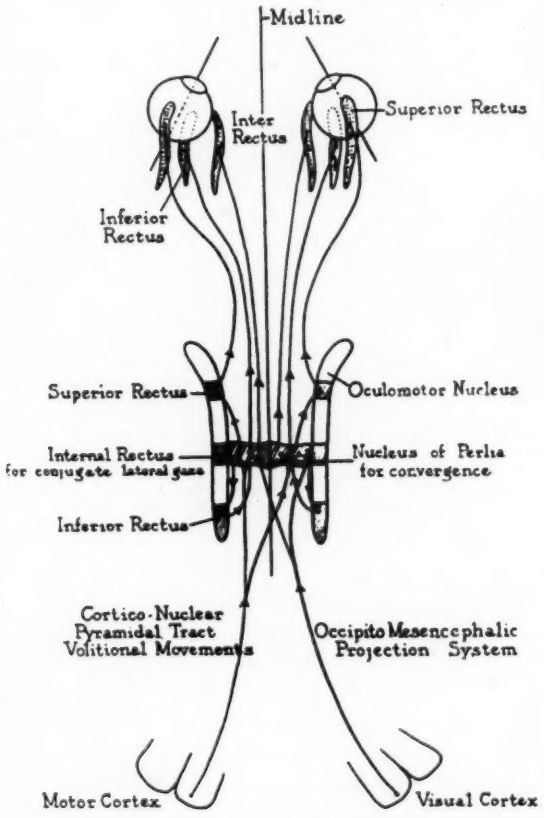

Fig. 4.—The mechanism for convergence (after Riley [72]).

Then, as the extensive developments and alterations took place in the efferent and afferent tracts [4] stereoscopic vision became automatic, and the individual, delivered from the necessity of concentrating on the effort of seeing, was free to devote his attention to the object seen.[6] All this was associated with enormous enhancement of the perceptive,

5. It really exemplifies a sort of phylogenetic shortcut.

6. Smith, Elliot: Evolution of Man, New York, Oxford University Press, 1927.

dynamic and cognitive possibilities of the human race.[7] Ontogenetically, man has now reached at least the second month of life.[8]

It is now possible to form some idea of the appearance of divergence in the phyletic scale. The nucleus of Perlia (fig. 4) is the last known nucleus to differentiate,[9] and its appearance in primates coincides with the appearance of the function of convergence. It may thus be assumed that divergence appears at this time and occupies an equally high and recent position on the phyletic scale.

This evolutionary review, brief as it is, has indicated how nature has been preparing the way for the highly specialized function of fusion. When any characteristic has been of such a nature as to further fusion it has been accentuated; when it would tend to render fusion more difficult it has been suppressed. As a result, the race has advanced tremendously in its mental and physiologic possibilities. The importance of fusion, attested by its late appearance in the phylum, is further proved by the solicitude concerning this function still shown by nature. All the other ocular functions are subservient to the maintenance of fusion,[10] and under certain conditions vision itself may be sacrificed in order to avoid diplopia. True fusion connotes the ability to focus the visual axes on points at varying distances, and the function of divergence is the sine qua non of this ability. If fusion is looked on as a chain forged by nature through the ages, the function of divergence must be considered one of its essential links.[11] The importance of this concept will become more evident as the thesis is developed.

Just as this exquisitely coordinated muscular structure has developed, so has man, by a process almost as evolutionary, increased his knowledge of that structure. As the enormous amount of the literature on the subject is reviewed it is found that an important stage of this knowledge

7. Schäfer, E. A.: Text-Book of Physiology, Edinburgh, Pentland, 1900, vol. 2, p. 1132.

8. Preyer, W.: The Senses and the Will, New York, D. Appleton and Company, 1893. Of course these theories are inexplicable unless one accepts the forbidden doctrine of the transmission of acquired characteristics. One is forced to accept the transmission of "biologically advantageous" characteristics if the thesis is to stand. It is curious that Collins, Wood-Jones and Smith seem unaware of their heresy.

9. Brouwer, B.: Klinisch-anatomische Untersuchung über den Oculomotoriuskern, Ztschr. f. d. ges. Neurol. u. Psychiat. **40**:152, 1918.

10. The exceptions to this rule, as described by Helmholtz (Physiologische Optik, ed. 2, Leipzig, Leopold Voss, 1896, p. 631), are based on the ability of the eye to make *exceptional* movements and need not apply in this case.

11. It may be objected that not fusion but the differentiation of perceptive patterns is the last link of this evolutionary chain. This, however, is higher than it is necessary to go to establish the point. The addition of an extra link to a chain is no argument against the existence of the other links; in fact, it implies their presence!

may be said to have fallen into each of three great periods. In the first (of which the earliest contribution [12] to be found is dated 1583) the chief attention was paid to the position of the eyes, and the oculist believed that if he had observed that the eye turned up, down or out he had done his duty, and that if he had added the learned diagnosis "luscitas" he had put the finishing touches on a masterly piece of work. Certainly, the conception of a pathologic state of the muscles as a disordered physiologic condition seems not to have entered the minds of the investigators in this period, and the literature is full of references to "myotomy" and "strabotomy."

The second period, which began about the middle of the nineteenth century, was initiated by von Graefe,[13] who began to direct attention to the individual ocular muscles. Many of his theories are now believed to be erroneous, but he contributed the knowledge of a new angle of approach to be used in diagnosis. This period is also marked by the work of Donders,[14] who was the first to note the connection between refraction and the muscular activities of the eye.

The third period began in the last decade of the nineteenth century and was dominated by Duane [15] until his death in 1926. It was he who classified the motor anomalies of the eye according to their etiology and who put the knowledge of ocular muscles on its present foundation. His nomenclature and methods of examination have been almost universally adopted ever since they were first described more than thirty-five years ago.[16]

DIVERGENCE

Divergence was defined by Duane as that function by virtue of which the visual lines, when converged on a near object, are made to separate in order to sight an object more remote. Attention will be

12. Bartisch, G.: Augendienst, Frankfort-on-Main, S. Feyrabend, 1583.

13. von Graefe, Albrecht: (*a*) Beiträge zur Physiologie und Pathologie der schiefen Augenmuskeln, Arch. f. Ophth. **1**:1, 1854; (*b*) ibid. **10**:156, 1864.

14. Donders, F. C.: Zur Pathogenie des Schielens, Arch. f. Ophth. **9**:1, 1863; Accommodation and Refraction of the Eye, London, Sydenham Society, 1864, p. 291.

15. Duane, A.: (*a*) Muscular Anomalies of the Eye, New York, J. A. Vail & Co., 1897; (*b*) A Case of Divergence Paralysis and Its Bearing upon the Theory of Squint and Heterophoria, Arch. Ophth. **28**:261, 1899; (*c*) in Fuchs, E.: Text-Book of Ophthalmology, Philadelphia, J. B. Lippincott Company, 1924, p. 267; (*d*) Associated Movements of the Eyes; Their Nerve Centers, Conducting Paths, Production Varieties and Derangements, Am. J. Ophth. **7**:16, 1924; (*e*) Monocular Movements, Arch. Ophth. **8**:530 (Oct.) 1932.

16. It has been deemed advisable to confine this grouping to contributors whose interest was mainly ophthalmologic and to exclude (perhaps unfairly) the numerous anatomists, physiologists and pathologists who have in the course of their work thrown much light on knowledge of the ocular musculature. To have included these would have been too great an undertaking for the scope of this work.

confined to lateral divergence, which for practical purposes is covered by this definition. It will be sufficient merely to allude to the vertical movements, defined as sursumvergence (up) and deorsumvergence (down). It will not be necessary to mention divergence of the vertical meridian, the visual lines remaining parallel.

In carrying out divergence the healthy eyes will, with unerring precision, fix the object sought. Diplopia must be accepted as the sine qua non of ocular paralysis in a previously binocularly sighted person.[17] It will be seen later that in paralysis of divergence diplopia supervenes, and it is therefore necessary to look on the function of divergence as an essential agency in maintaining fusion. Without divergence fusion is lost. This has already been mentioned and will be elaborated on later.

The question now arises whether divergence is an active process, carried out by active contraction of the abduction muscles, or whether it is a passive function, depending for its action on relaxation of the internal rectus muscles. If the latter view were adopted, it would follow that the natural position of the eyes in complete muscular relaxation would be one of divergence of a greater or less degree. The investigators [18] of this theory seemed unaware of some of the pathologic possibilities in the function of divergence and were almost certainly basing their opinions on some abnormal cases. In favor of divergence as an active process, it may be noted that a person whose divergence is being tested with prisms base in experiences the same sense of strain experienced in overcoming a prism base out (in the latter case convergence, an admittedly active factor, is being exercised). Against this may be placed the fact that the divergence power, as measured by prisms base in, is constant and cannot be increased by exercise beyond the original amount.[19] If the eyes diverged solely as a result of the elasticity of the external rectus muscles, the divergence should be more rapid at the beginning than at the end of divergence, the external rectus muscles being then under more tension. Such cannot be the case, because divergence is carried out at uniform speed; and so it must be assumed that an active factor enters into the mechanism as a reenforcement. Furthermore, in cases of paralysis of divergence (which will be discussed

17. This assumes that the muscles at fault are not associated antagonists, such as, for instance, the superior rectus and the contralateral inferior oblique, affected to the same degree. This is hair-splitting and is mentioned as a remote possibility.

18. Grut, Hansen: Bowman Lecture, Ophth. Rev. **10**:16, 1890. Leconte, J.: Sight; An Exposition of the Principles of Monocular and Binocular Vision, New York, D. Appleton and Company, 1881.

19. One must agree with Landolt (Landolt, E., in Norris, W. F., and Oliver, C. A.: System of Diseases of the Eye, Philadelphia, J. B. Lippincott Company, 1900, vol. 4, p. 77) that this is not a strong argument, but one feels that some of his assertions supporting the contrary view are equally unsound.

later) the initial movements are but little affected, while the later ones, i. e., those produced as the eye approaches increasingly distant points of fixation, are increasingly interfered with. In cases of complete paralysis of the external rectus muscle the eye cannot diverge as far as the midline when an attempt is made to rotate the globe toward the field of the paralyzed muscle, even when the internal rectus muscle is tenotomized. This certainly indicates that the normal action of the external rectus muscle does not cease at the midline but is carried for some distance to the inner side.[20]

The experimental work is also somewhat conflicting. Sherrington [21] cut the oculomotor and trochlear nerves and stimulated the cortex on the same side. He found that the external rectus muscle on the other side contracted and that the eye in which the third and fourth nerves had been cut moved to the midline but no farther. Fitting this into correlation with other findings, he formulated his "theory of reciprocal innervation," which presumed the active relaxation of an anatomic antagonist when a given muscle is in contraction. If, however, it is assumed that the ocular muscles are similar physiologically to those of the rest of the body (and there seems to be no good reason for assuming otherwise), a somewhat different conception is found to arise on the basis of the work of Tilney and Pike.[22] These investigators, in studying the synergic action of the cerebellum, found that in every movement the dominant muscle is checked by an exactly controlled simultaneous contraction of its antagonist.[23] This synergism is responsible for the exquisite accuracy of muscular coordination. From a consideration of the function of divergence one would be tempted to adopt this view and to assume that tonic changes take place in all the muscles involved in synergic movements.

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20. Mauthner's (Mauthner, L.: Die nicht nuklearen Lähmungen, Vortr. a. d. Ges.-Geb. d. Augenh. **2**:293, 1886) explanation of this as a loss of muscular tone contributes nothing, muscular tone being merely residual muscular contraction. Similarly, Berry's (A Case of So-Called Divergence Paralysis, Tr. Ophth. Soc. U. Kingdom **21**:268, 1901) elaborate explanation, when critically examined, resolves itself into being merely an inflation of the "divergence by elasticity" theory. This has already been dealt with.

21. Sherrington, C.: Experimental Notes on Two Movements of the Eye, J. Physiol. **17**:1994, 1927; Proc. Roy. Soc., London **64**:120, 1898.

22. Tilney, F., and Pike, F. H.: Muscular Coordination Experimentally Studied in Its Relation to the Cerebellum, Arch. Neurol. & Psychiat. **13**:289 (March) 1925.

23. This does not mean that the existence of cocontraction in a pair of antagonistic muscles disproves the law of reciprocal innervation (Adler, F. H.: Reciprocal Innervation of the Extraocular Muscles, Arch. Ophth. **3**:318 [March] 1930). It does, however, throw some doubt on Sherrington's conception of the application of that innervation.

How is one to evaluate these conflicting opinions? It must be remembered that the earlier investigators assumed that the function has to be either active or passive. Duane [15b,e] held the function to be both active and passive, assigning the passive rôle to the internal rectus muscles and an entirely active part to the external rectus muscles. This view, although it fits in well with Sherrington's theory of reciprocal innervation, is not compatible with the opinion held by Tilney and Pike. However, it may well be that there is a passive factor in the act, but that it should be assigned to the external rather than the internal rectus. If one adopts this point of view, one can still imagine the eye diverging by both active and passive means, each action following and blending with the preceding action but having its *point d'appui* in the external rectus muscles. As divergence begins, the external rectus muscles, being stretched, do not contract actively, the passive external rotation being checked by finely graded tonic movements in the internal rectus muscles. As divergence proceeds, the external rectus muscles are brought into active contraction to an increasing degree.[24] The major part of the act is therefore active rather than passive.

The question of voluntary divergence naturally arises at this stage. A careful distinction must be made between voluntary abduction of both eyes (which would be true voluntary divergence) and voluntary abduction of one eye, the other remaining fixed. No cases of the former condition can be authenticated, and the function may be presumed not to exist. The second class is well represented and is not uncommon.[25] How, then, is this phenomenon to be reconciled to Hering's law? [26] The answer would be easier to supply if many of the authors had attempted to rule out the presence of a divergence excess which will be discussed later. I have seen two cases of voluntary abduction in apparently normal persons who were found on examination to be the unsuspecting possessors of excessive divergence. They were obviously relaxing their accommodation and convergence and thus allowing their eyes to return to what was for them the position of rest. These cases, since an

24. The strongest proof of this contention lies in the phenomenon known as "amplitude of recession," which will be discussed later under "Pathology." This important sign, much neglected by ophthalmologists and apparently unknown to neurologists, offers a solution to many puzzling problems arising from the investigation.

25. Wilbrand, H., and Saenger, A.: Neurologie des Auges, Munich, J. F. Bergmann, 1921, vol. 8, p. 17. Spiller, W. G.: Unilateral (Unassociated) Innervation of the Ocular Muscles, Arch. Neurol. & Psychiat. **18**:691 (Nov.) 1927. Reese, W. S.: Disjunctive Ocular Movements, Arch. Ophth. **10**:564 (Oct.) 1933; ibid., discussion by Langdon and Adler.

26. This law is to the effect that cortical innervation of ocular muscles is never unilateral but that proportionate innervation takes place in all the movements initiated by the cortex.

abnormality was present, threw no definite doubt on the applicability to Hering's law, which applies to physiologic, not pathologic, functions. Nevertheless, patients with a similar condition have been described as being normal in all other respects. In this connection it must be remembered that voluntary unilateral movement is certainly present in some of the lower animals, and it may be that such an occurrence in man is indicative of potential cortical control and is a manifestation of a reversion to a more primitive type of movement.

Divergence, then, while partially an active process, is not a volitional function. It is obvious, however, that it is dependent on volitional impulses for its initiation. What, then, is the motivating force, the *deus ex machina,* of the act? It is impossible to be dogmatic in the present state of knowledge. To discuss the matter fully would involve taking into account the sense of position of the eyes and would draw in spatial considerations which are beyond the scope of this article.[27]

As early as 1880, Hering [28] noted and discussed the "faculty of attention." Before the eyes begin to move the attention is occupied by the point in space aimed at, and the initiatory, regulatory and terminative impulses of divergence owe their origin to this highly situated faculty. It may be that the explanation of the motivation in divergence lies in that direction. Certainly, in the light of this theory, Köllner's [29] article (in which he assigns a position sense for the eyes to a subcortical center) should be read with renewed interest.

The avenues connecting these higher centers with the lower ones are not well known. However, as lesions of the posterior longitudinal bundle do not cause paralysis of divergence and because this function is not interfered with in unilateral cortical or subcortical lesions, it may be deduced that these fibers do not pass through the fasciculus (as do most other association fibers) and that the cerebral centers, as well as the descending paths, must be bilateral.[30] This will have an important bearing on the subject when the time comes to consider the question of a center for divergence.

II. PATHOLOGY

Hitherto the discussion has been confined to conditions arising from normal function of the eyes, and it has been necessary to reason a priori.

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27. Bourdon: La perception visuelle de l'espace, Paris, Schleicher Bros., 1902, p. 66. Marx: Ztschr. f. Sinnesphysiol. **47**:79, 1915. Tschermak: Ergebn. d. Physiol. **188**:222, 1921.

28. Hering, in Hermann, L.: Handbuch der Physiologie, Berlin, A. Hirschwald, 1880, p. 534.

29. Köllner: Klin. Wchnschr. **2**:482 and 1293, 1923; Arch. f. Augenh. **93**:130, 1923.

30. Duane [15d] discussed clinical and other proof.

From time to time, however, references to pathologic conditions have crept in. The evidence afforded by diseased conditions is most important.

Anomalies of divergence fall into three groups: (1) paralysis, (2) hypokinesis (divergence weakness) and (3) hyperkinesis (divergence excess).

PARALYSIS OF DIVERGENCE

Cases of paralysis of divergence have been known for a long time, although they have not always been recognized as such. For instance, von Graefe,[13b] whatever his ideas of etiology were, observed the condition as early as 1864, and a case seen by Priestley Smith [31] in 1875 and reported with others four years later certainly fell into this category.[32] It was Parinaud,[33] however, who first evaluated the syndrome correctly, in 1883; and it is to this observer that priority is universally given in the literature. Duane's [15b] classic description in 1899 finally put the subject on a sound basis. Numerous cases have been reported, the most accurate contributions coming from the pens of Hoffman,[34] Bielschowsky,[35a] Straub,[36] Popow,[37] Braunschweig,[38] Dor,[39] Flesch,[40] Zentmayer,[41] von Hippel,[42] Wheeler,[43] Dunnington,[44] Alger,[45] Holden,[46] Lebensohn,[47] Dunphy,[48] Howard,[49] and Stokes.[50]

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31. Smith, Priestley: Ophth. Hosp. Rep. **9**:22, 1879.
32. Smith did not diagnose the condition as paralysis of divergence, however.
33. Parinaud, H.: Arch. de neurol. **5**:14 and 145, 1883.
34. Hoffman: Ber. u. d. Versamml. d. ophth. Gesellsch. z. Heidelberg **1**:117, 1900.
35. Bielschowsky, A.: (*a*) Ber. u. d. Versamml. d. ophth. Gesellsch. z. Heidelberg **1**:110, 1900; (*b*) Divergence Excess, Arch. Ophth. **12**:157 (Aug.) 1934.
36. Straub, M.: Centralbl. f. prakt. Augenh. **21**:8, 1897.
37. Popow: Neurol. vestnik **11**:1, 1903.
38. Braunschweig, W.: Klin. Monatsbl. f. Augenh. **46**:644, 1911.
39. Dor, L.: Ophth. Klin. **2**:14, 1898.
40. Flesch: Ophth. Klin. **9**:23, 1905.
41. Zentmayer, W.: Ophth. Rec. **22**:373, 1913.
42. von Hippel, A.: München. med. Wchnschr. **27**:122, 1902.
43. Wheeler, J. M.: Canad. M. Quart. **4**:273, 1919.
44. Dunnington, J. H.: (*a*) Paralysis of Divergence with Report of Cases Due to Epidemic Encephalitis, Arch. Ophth. **52**:39, 1923; (*b*) Divergence Excess: Its Diagnosis and Treatment, ibid. **56**:344, 1927; (*c*) personal communication to the author.
45. Alger, E.: Tr. Am. Ophth. Soc. **14**:665, 1914.
46. Holden, W.: Arch. Ophth. **1**:101, 1929.
47. Lebensohn, J.: Paralysis of Divergence with Chorea and with Tabes, Am. J. Ophth. **9**:684, 1926.
48. Dunphy, E.: Paralysis of Divergence, Am. J. Ophth. **11**:298, 1928.
49. Howard, H.: Divergence Paralysis, Am. J. Ophth. **14**:736, 1931.
50. Stokes, W. H.: Paralysis of Divergence, Arch. Ophth. **11**:651 (April) 1934.

In reviewing the literature one is struck by the similarity in symptomatology in all the cases reported. The cardinal symptoms are homonymous diplopia and convergent strabismus of sudden onset (fig. 5). The report of a typical case follows. This case was selected from the mass of available material chiefly because the record [44a] is complete.

The patient woke up seeing double. The findings are listed as follows: homonymous diplopia; esotropia, with the right eye fixing at 6 meters, 35△; esophoria at 30 cm., 6△; near point of convergence, 80 mm.; binocular single vision by approximation [51] at 40 cm.; binocular single vision by recession [51] at 50 cm.; no

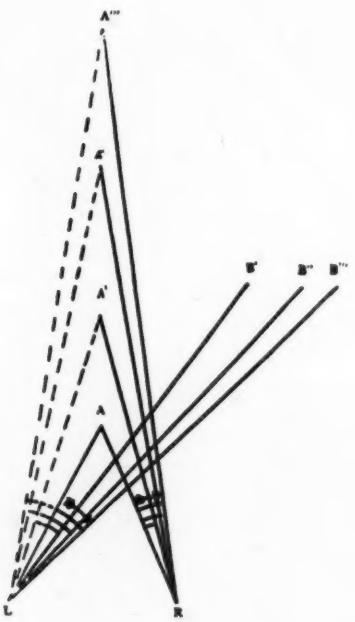

Fig. 5.—Paralysis of divergence. The paralysis is such that both eyes naturally tend to converge so as to be directed at the point *A*. When the attempt is made to sight the point *A'* farther off from the eyes, the eyes cannot, as they would naturally do, move simultaneously outward through the equal angles *ALA'* and *ARA'*. The only way in which one of the eyes can sight the point *A'* is for that eye to move outward while the other eye moves inward to an equal extent. This it can do, since the power to perform associated lateral movements is unimpaired. Hence as *R* moves out through the angle *ARA'*, *L* moves inward through the equal angle *ALB'*. Hence it is directed at *B'* instead of *A'*, so that the deviation is represented by the angle *A'LB'*, or twice *ARA'*. So, as the right eye turns still farther out to *A''*, *A'''*, etc., the left eye turns still farther in to *B''*, *B'''*, etc. (After Duane.[15b])

51. This sign has been referred to before. As the object is approximated to the eyes a point of binocular single vision is found—in this case at 40 cm. As the object recedes binocular vision is not lost at once but is retained—in this case for 10 cm.

increase in diplopia in any field; no limitation of motion of either eye in any field, and accommodation of 4 diopters in the right eye and 3.5 diopters in the left.

The symptomatology in all cases follows this model closely.

The etiology is varied, diseases of the brain being at fault in nearly every case. The condition has been ascribed to encephalitis,[52] syphilis, tabes,[15b] multiple sclerosis,[53] lead poisoning,[15b] hemorrhage,[54] hysteria,[42] diphtheria,[45] poliomyelitis,[55] chorea,[47] and metastatic carcinoma.[49] Tumor of the brain was suggested as a cause by Duane,[15b] but on insufficient pathologic evidence. Berry's [56] theory (that the condition is due to a spasm of convergence) has been discredited by more recent additions to the knowledge of this subject and needs only to be mentioned.

The diagnosis is made on the basis of the sudden onset, the concomitant type of the diplopia and the periodicity of the esotropia. The differential diagnosis is chiefly concerned with bilateral paralysis of the sixth nerve. In cases of paralysis of the abducens nerve the plotting of the diplopia is the most valuable aid, as the images will be found to separate in the fields of lateral gaze. In old cases there is also limitation of movement in the eyes to the right and left.

Spasm of convergence may also be confused with paralysis of divergence. If the diplopia or deviation is due to convergence excess, it increases as the test object is brought closer to the eyes and diminishes as it recedes. Differentiation of paralysis of divergence from other anomalies of divergence will be taken up later.

The treatment is at first conservative, consisting of occluding an eye to get rid of the diplopia. Training by prisms base out, which is sometimes suggested as a means of relief, is not practicable, on account of the large amount of correction needed to overcome the deviation and on account of the periodicity of the squint. Exercises are of no avail. If the position has become stationary (after the lapse of at least a year), an operation should be performed. Resection or advancement of one or both rectus muscles would seem to be the logical procedure.

In the matter of prognosis it is impossible to be categorical. Recession is frequent; conditions due to diphtheria have the best outlook, those due to encephalitis the worst. A high amplitude of recession may

52. Zentmayer, W.: Ophth. Rec. **22**:373, 1913. Dunnington, J. H.: Divergence Excess: Its Diagnosis and Treatment, Arch. Ophth. **56**:344 (July) 1927. Holden.[46]

53. Duane, A.: A Case of Divergence Paralysis and Its Bearing upon the Theory of Squint and Heterophoria, Arch. Ophth. **28**:261, 1899. Dunphy, E.: Paralysis of Divergence, Am. J. Ophth. **11**:298, 1928.

54. Wheeler.[43] Alger, E.: Tr. Am. Ophth. Soc. **14**:665, 1914.

55. Cutler, in discussion on Wheeler, J. M.: Case of Divergence Paralysis, Arch. Ophth. **47**:419, 1922.

56. Berry, G.: A Case of So-Called Divergence Paralysis, Tr. Ophth. Soc. U. Kingdom **21**:268, 1901.

indicate a better prognosis,[44c] but there is no proof of this as yet. It should be emphasized at this point that paralysis of divergence usually goes on to paralysis of the external rectus muscles. This important fact will be mentioned again.

DIVERGENCE INSUFFICIENCY

Insufficiency of divergence may be present as weakness rather than as paralysis of the function. It does not have its origin in underdevelopment of the muscles themselves (structural heterophoria) or in variations in the origin or insertion of the muscles (insertional heterophoria).[57] Cases in which there is actual weakness of the external rectus muscles cannot be classed as cases of true weakness of divergence. The latter may be primary, but in the majority of the cases the condition is due to progressive convergence excess.

The findings are: For distance there is esophoria, varying in amount from 2 to 8 prism diopters in all tests. All associated lateral movements are normal. Prism divergence is greatly reduced and is usually in direct proportion to the esophoria. Thus, in a typical case showing 3 prism diopters of esophoria, the divergence power was 4 prism diopters; and in another, with esophoria of 5 prism diopters the diverging power was 0.5 prism diopter. In the idiopathic cases there is orthophoria or even exophoria for near objects. If the condition is secondary to convergence excess, the esophoria for near objects is greater than for distance and is disproportionately large when compared with the deficiency in divergence power.

The symptoms are troublesome. There are marked asthenopia, often of the "panoramic" type,[58] headache and lassitude. Near work does not usually produce an increase in the symptoms, unless the divergence weakness is associated with a simultaneous anomaly of convergence. Intermittent spontaneous diplopia may occur. It is homonymous and periodic.

In the differential diagnosis the chief distinction has to be made from convergence excess. The salient features will be set forth later in tabular form.

The treatment of divergence insufficiency is difficult. Duane[15b] reported failure to improve the condition by means of exercises with prisms and cautioned against the constant wearing of prisms base out, this being productive of further enfeeblement of the abducting power.

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57. Schneller: Anatomisch-physiologische Untersuchungen über die Augenmuskeln Neugeborener, Arch. f. Ophth. **47**:178, 1898. Weiss, L.: Arch. f. Augenh. **33**:29, 1896; condensed translation, Arch. Ophth. **25**:341, 1896.

58. This is a headache initiated or aggravated by observing distant objects, such as processions or athletic contests, or by watching the landscape from a train window.

From a practical point of view, however, such generalizations are not accurate; and so, if there is no convergence excess, resection of one or both external rectus muscles should be performed.

The prognosis in untreated cases depends on the appearance of convergence excess. The idiopathic variety of divergence insufficiency remains stationary, while the variety associated with spasm of convergence tends to progress, and convergent strabismus may eventually supervene.

DIVERGENCE EXCESS

The anomalies of divergence already discussed have had to do with underexercise of the function. The condition in which one finds hyperkinesis of divergence is known as divergence excess.

This condition was known to von Graefe [13] as early as 1854 and was described more fully by Alfred Graefe [59] in 1898.[60]

Abnormalities of structure and insertion are not found, and the idiopathic variety is a true innervational anomaly of the hyperkinetic type.[61] The secondary type is a sequela of convergence weakness. The condition is fairly common, occurring in from 1 to 2 per cent of eleven thousand patients examined by Berens and his collaborators [62] in New York.

The etiology is obscure. Wootton [63a] and Young [64] expressed the belief that refraction plays a part, but this was denied by Dunnington [44b] and Berens and his collaborators [62] and was considered doubtful by Duane [15a] and Maxwell.[65]

The symptoms are frequently slight, and the patient sometimes has no complaints, provided that the mechanism of convergence is unaffected and a state of vertical orthophoria exists. There may be intermittent transient exotropia attended by crossed diplopia, which is easily overcome, as a rule, by voluntary convergence effort. When symptoms are present they consist of headache and asthenopia of the panoramic type.

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59. Graefe, Alfred, in von Graefe, A., and Saemisch, E. T.: Handbuch der gesammten Augenheilkunde, Berlin, Julius Springer, 1898, vol. 8, pt. 1.

60. Duane [15a] had already (1897) put it on a sound basis as regards diagnosis and nomenclature.

61. Bielschowsky [35b] expressed the belief that divergence for distance may be the normal position of rest in certain persons. Nevertheless, if a patient has excessive divergence he has divergence excess.

62. Berens, C.; Hardy, L., and Stark, E.: Divergence Excess: Its Incidence; Its Correction with Refraction; Value of Orthoptic Treatment, Tr. Am. Ophth. Soc. **27**:263, 1929.

63. Wootton, H.: (*a*) Ophthalmoscope **14**:236, 1916; (*b*) Arch. Ophth. **44**: 440, 1925; (*c*) New York State J. Med. **15**:56, 1915.

64. Young, C. A.: Virginia M. Monthly **53**:25, 1926.

65. Maxwell, E.: Brit. J. Ophth. **3**:340 and 408, 1919.

The signs are:

1. For distance there is exophoria of from 5 to 30 prism diopters or more. The power of divergence, as measured by the prism base out, is greatly increased and may be as high as 33 prism diopters (Dunnington).[44b] It is a constant factor, unvarying throughout repeated examinations. Lateral movements of the eyes are unaffected. There may be intermittent crossed diplopia, which is usually superable. This does not vary in amount in the lateral fields [66] but diminishes as the test object is approximated to the patient.

Distinguishing Signs of Convergence Excess, Divergence Insufficiency, Convergence Insufficiency and Divergence Excess

Nature of Test	Convergence Excess	Divergence Insufficiency	Convergence Insufficiency	Divergence Excess
For distance all tests (screen, parallax, Maddox rod, phorometer) show	Orthophoria or slight esophoria	Esophoria greater than normal	Orthophoria or slight exophoria	Exophoria greater than normal
For near vision all tests show	Esophoria greater than normal (much more than for distance)	Esophoria slight or none (decidedly less than for distance)	Exophoria much greater than normal (much more than for distance)	Exophoria decidedly less than for distance
Near point of convergence	Normal (or abnormally close)	Normal	Remote (more than 9 cm. from base line connecting centers of eyes)	Normal
Prism convergence	Normal	Normal	Low and accomplished only with effort and difficulty	Normal
Prism divergence	Normal	Low (less than 1-1.5 P.D.) or absent (i. e., insuperable homonymous diplopia for distance)	Normal	Abnormally great (more than 4.5 P.D.)
Primary and secondary screen deviation	In all types deviation of one eye behind the screen, measured with prisms, equals precisely that of the other eye (distinction from nonconcomitant esotropia)		In all types deviation of one eye behind the screen equals precisely that of the other eye (distinction from nonconcomitant exotropia)	

2. For near objects muscular deviation is within normal limits, and the near point of convergence *(PcB)* is normal.

In the differential diagnosis the chief condition to be ruled out is convergence insufficiency. The important distinguishing signs are set forth in the table (modified from that of Fuchs).[15c]

A less difficult differentiation is to be made from weakness of an adductor muscle. In this condition the exophoria and diplopia, whether for distance or near objects, increase as the eye is brought into the field of the affected muscle.

66. It increases in the upper fields and diminishes in the lower, but this is a characteristic of any form of crossed diplopia.

The course is slowly progressive, often terminating in divergent strabismus. This may persist for a while only for distance, but gradually the convergence weakens and the deviation becomes permanent.

The method of treatment to be adopted has long been a bone of contention among ophthalmologists. The choice lies between orthoptic training by prisms and operation. The opinion that prisms are valuable was expressed by Berens and his collaborators,[62] Reber,[67] Young,[64] Bulson [68] and Allen.[69] Maxwell [65] expressed the view that they are frequently of value. Duane [15c] stated that they are occasionally valuable. Wootton [63] asserted that they are of slight value only, and Dunnington [44b] and Lauder,[70] that they are of no use.

What is the explanation for these widely differing convictions held by competent observers? In seeking an answer to this question, one finds that the disputants are not on common ground. Selecting the most articulate representative in each camp, one discovers that both Dunnington [44b] and Berens and his collaborators [62] agree as to the findings for distance in divergence excess but differ in their ideas as to what the finding for near vision should be. Dunnington [44b] stated: "The amount of deviation at 13 inches should be within the normal limits, i. e., it would range anywhere from orthophoria to 4° of exophoria . . . Any abnormality of the findings at this distance denotes an anomaly of convergence."

A study of the cases described by Berens and his collaborators [62] discloses that the findings for near vision vary from an exotropia of 16 prism diopters to an exophoria of 10 prism diopters and that the average of the exophoria for near vision is 20 prism diopters. In four of these cases secondary convergence insufficiency was also diagnosed, and in the cases in which a diagnosis of straightforward divergence excess was made there was exophoria for near objects amounting to 10, 30 and 18 diopters, respectively. If one accepts Dunnington's view—and it must be conceded that, by definition, it is the correct one—one must conclude that Berens and his collaborators were treating the convergence insufficiency. It appears, then, that pure divergence excess is not amenable to orthoptic training but that in cases in which convergence is affected the condition may be expected to yield to this measure.

Patients who have been treated conservatively without avail should be operated on. Open tenotomy of one or both external rectus muscles may be done, depending on the amount of deviation for near vision.

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67. Reber, W.: Ophthalmoscope **12**:456, 592 and 644, 1914.
68. Bulson, A.: J. Indiana M. A. **19**:107, 1926.
69. Allen, S.: New York State J. Med. **15**:60, 1915.
70. Lauder, E.: Cleveland M. J. **9**:768, 1910.

Overcorrection of from 10 to 15 prism diopters should be sought. Any convergence weakness should then yield to treatment with exercises.[71]

Primary divergence excess may increase in amount or may persist unchanged for years. The exophoria may gradually become exotropia—at first for distance and, later, as convergence weakens, for near objects as well. The end-result may thus be concomitant divergent strabismus.

THE CENTER FOR DIVERGENCE

A center is defined [72] as "the Ganglion or plexus whence issue the nerves controlling a function." In discussing the existence of a center for divergence it must be admitted that one is in no position to be categorical and that reasoning will have to be guided by deduction rather than influenced by definite proof. It must be considered axiomatic that no function of any importance is without its center; and therefore, if it can be established that divergence as a function is sufficiently important, one will be justified in assuming the existence of a center for divergence. But first it is necessary to prove that divergence is a function in its own right and not a merely incidental movement in the complex ocular mechanism.

Given an eye in a state of convergence, it is obvious that lateral divergence must be carried out by (1) elasticity and/or (2) active contraction of the external rectus muscles.

The rôles played by these processes have already been discussed, and it has been found that the major part is played by the latter of the two possibilities. It must now be conceded that active contraction of the external rectus muscles can take place only by means of innervation *(a)* through the abducens nuclei or *(b)* from some other source.

It has already been proved that in pure paralysis of divergence the abducens nuclei are intact, and one is therefore forced to the conclusion that divergence must be carried out through some other source. It cannot be said yet, however, that the function arising from this source is exclusively one of divergence unless it can be proved that the function is associated solely with the act. It seems that the numerous cases in which divergence and divergence alone is affected furnish sufficient evidence to prove this contention. It is safe to assume at this point that the faculty of divergence is a function per se. Having established this, it remains to be decided whether the movements involved are suffi-

71. If the primary condition were convergence insufficiency, one would naturally correct the convergence weakness first and then, if necessary, correct the divergence excess.

72. Gould, G.: Medical Dictionary, ed. 3, Philadelphia, P. Blakiston's Son & Co., 1931, p. 288.

ciently intricate to lie beyond the capacity of noncentral control, or, to put it otherwise, whether they are important enough to merit a center of their own.[73]

Nature has devoted her attention through the ages to building up a function of binocular stereoscopic vision which depends for its maintenance on the integrity of the fusion faculty. Coincident with the acquisition of convergence, divergence must have been acquired; otherwise, fusion would have been impossible. In other words, divergence is an integral part of the chain that nature, in spite of all difficulties, has succeeded in forging. It is inconceivable that she would leave uncontrolled by a center this link of her phylogenetic chain, nullifying by this means the evolutionary advance of ages. As she developed centers, she invariably developed antagonistic centers. The existence of a center for convergence (fig. 4) [74] is well authenticated. It is most improbable that nature, contrary to her rule and against her own interests, should leave this important and powerful center without its physiologic antagonist. The movements concerned in divergence require the same delicacy of adjustment and a precision of control required in carrying out the functions of convergence and associated lateral movements. These functions have authentic centers (figs. 6 and 7),[74] and it is reasonable to believe that the analogous function of divergence has a center as well.

The evidence derived from pathologic study is more conclusive.

The function of divergence arises from bilateral innervation. Now, paralysis of divergence (which must be accepted as a clinical entity) could follow a lesion only in (1) the cortex, (2) the paths below the cortex or (3) a divergence nucleus.

A lesion in either or both of the first two situations would almost certainly have such a distribution as to produce paralyses of lateral movements on both sides and would almost as certainly be of such extent as to cause mental hebetude or even death. That such lesions may cause paralysis of divergence is certainly possible, but in such a case other symptoms must predominate. That such lesions may cause pure divergence paralysis is almost inconceivable. The only situation left to be occupied by a lesion that is to produce simple divergence paralysis is a divergence nucleus or center.

Reasoning in another way, let it be assumed (purely for the sake of argument and at some pains to avoid begging the question) that there

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73. The very words of the definition imply that if a function exists, there must be a center to control it (Charcot, quoted by Landolt, E., in Norris, W. F., and Oliver, C. A.: System of Diseases of the Eye, Philadelphia, J. B. Lippincott Company, 1900, vol. 4, p. 77).

74. Riley, H.: Central Nervous System Control of Ocular Movements and Disturbances of This Mechanism, Arch. Ophth. **4**:640, (Nov.); 885 (Dec.) 1930.

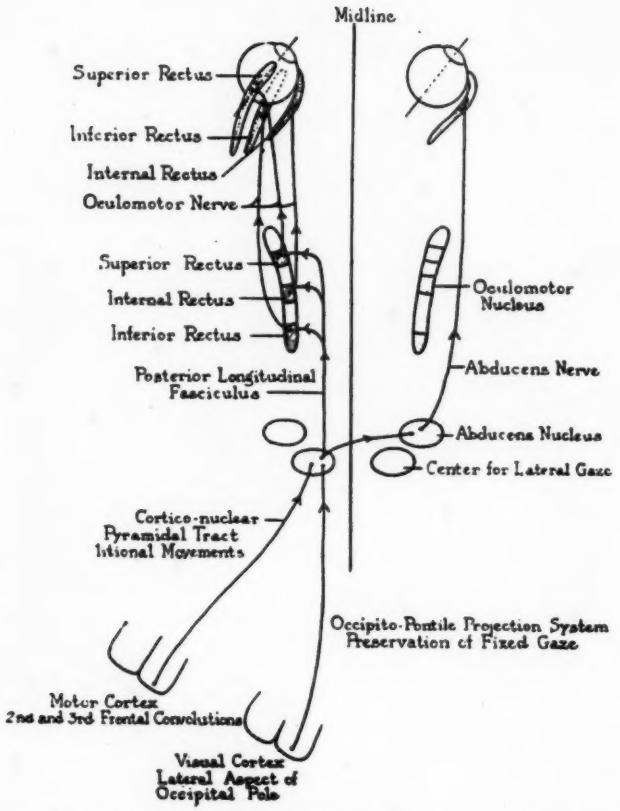

Fig. 6.—Mechanism for lateral deviation of gaze (after Riley [14]).

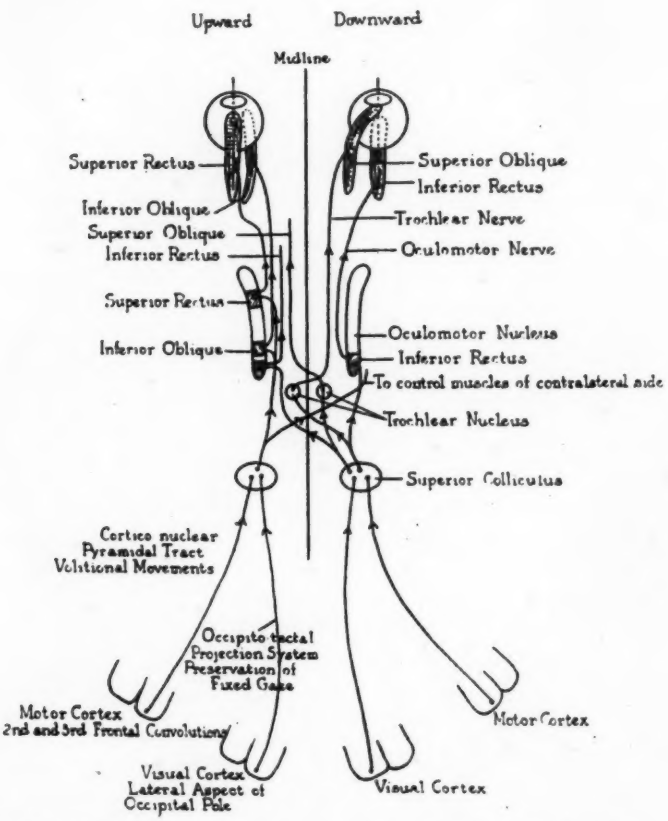

Fig. 7.—Mechanism for vertical deviation of gaze (after Riley [74]).

is a center for divergence. What, then, would be the clinical picture produced by a lesion in this situation alone? There would be no interference with the sixth nerve, because, by the premise, that nucleus is intact. Lateral movements would therefore be unimpaired. Convergence would be unaffected; but, since the center antagonistic to that of convergence is destroyed, the visual axes would be approximated. As the gaze was directed away, however, diplopia would supervene (fig. 5). Theoretically, all this would occur if it were assumed that a center for divergence existed and became paralyzed. The clinical picture so produced, however, would be exactly that of so-called paralysis of divergence!

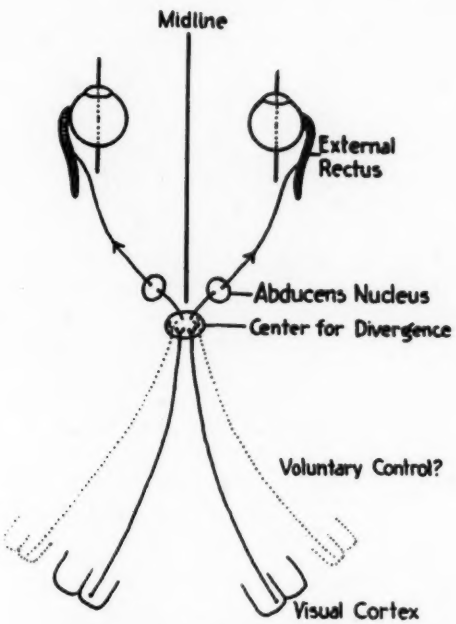

Fig. 8.—Center for divergence.

Certainly, the evidence on physiologic, phylogenetic and pathologic grounds seems, in the light of present knowledge, to establish the existence of a center for ocular divergence.[75]

75. It may be asked why, if a center for divergence really exists, *active* voluntary divergence is not possible. Turning again to a consideration of the antagonistic function, one finds that a person cannot rotate an eye so far nasally on convergence as he can on associated movements, because it is never necessary for him to focus on objects a few millimeters from his eyes. It would seem that the human race has not attained the faculty of active voluntary divergence for the same reason that the individual cannot perform the maneuver mentioned—there is not enough biologic advantage in such a function to justify its acquisition and development.

Having assumed that a center for divergence is present, where is it placed? Parinaud believed that it was in the cerebellum. His reasons for this belief are unconvincing in the light of later knowledge and will not be mentioned. In cases of pure paralysis of divergence, the abducens nucleus is unaffected; but later, particularly in cases of encephalitis, paralysis of divergence becomes true abducens nerve paralysis. From these facts [76] and from the assumption that the abducens nerve is the "pacemaker" of divergence, one may postulate the existence of a center for divergence separate from, but adjacent to, the nucleus of that nerve. Since the external rectus muscles are innervated only ipsilaterally, this nucleus would probably be in the midline (fig. 8).

76. Duane, A.: Monocular Movements, Arch. Ophth. **8**:530 (Oct.) 1932. Straub, M.: Centralbl. f. prakt. Augenh. **21**:8, 1897.

Correspondence

HANDBOOK OF OCULAR THERAPEUTICS: A CORRECTION

To the Editor:—A mistake in my "Handbook of Ocular Therapeutics" has come to my attention, and I should very much appreciate it if the following note of correction could be published in the ARCHIVES. On page 69, in the table for dosage of old tuberculin, doses nos. 33 to 36 are given as .2 to .5 cc. of concentrated old tuberculin. This would be taken to mean the original pure old tuberculin and would, of course, mean a much larger dose than the one desired. In cases which require doses over 1 mg., a special dilution must be made up containing 0.1 cc. of concentrated old tuberculin to 10 cc. of physiologic solution of sodium chloride. Of this, 0.1 cc. equals 1 mg. of tuberculin and doses nos. 33 and 36 should be 0.2 cc., 0.3 cc., 0.4 cc. and 0.5 cc. of this dilution. The other statements as to dosage are correct.

This error has been corrected in the second printing of the book.

SANFORD R. GIFFORD, M.D., Chicago.

News and Notes

GENERAL NEWS

Report on Sight of School Children.—A report entitled "Conserving the Sight of School Children," prepared under the direction of Dr. Thomas D. Wood of Columbia University and published by the National Society for the Prevention of Blindness, states that one eighth of all the school children in the United States are handicapped by defective eyesight. The three most common causes of defective vision are, in the order named: farsightedness, astigmatism and nearsightedness. In the final chapter the statement is made that one of every 500 children comes under the classification of "partially seeing." Sight-saving classes numbering 458, maintained by 145 communities, now have an enrolment of 6,000 children.

Examinations by American Board of Ophthalmology.—Examinations by the American Board of Ophthalmology will be held in Philadelphia on June 8 and in New York on June 10. Applications must be filed before April 10 and should be mailed to the secretary, Dr. William H. Wilder, 122 South Michigan Avenue, Chicago.

Intermediate Ophthalmic Service.—As a result of the activities of the Ophthalmological Society of New South Wales, the Medical Eye Service of New South Wales has been instituted for the benefit of persons of limited means. The affairs of the organization are controlled by the Ophthalmic Association, Limited, the shareholders of which are members of the Ophthalmological Society of New South Wales.

Consultation rooms, open to patients six days and five evenings a week, are located at 267 Elizabeth Street, Sydney. Persons eligible for attention at this institution are members of friendly societies and their families and persons receiving a salary of less than £260 yearly and their dependants.

SOCIETY NEWS

The Pan-American Medical Association.—On July 18, 1935, members of the Pan-American Medical Association and their families and friends will sail from New York on the S. S. *Columbia* to the medical convention in Rio de Janeiro, returning August 28. The itinerary is as follows: New York, Havana, Curaçao, five days at Rio de Janeiro, three days at Santos, Trinidad, Santo Domingo, Kingston (Jamaica), Havana and New York. Information about the cruise may be obtained from the Pan-American Medical Association, 745 Fifth Avenue, New York.

Tsinan Ophthalmological Society.—The Tsinan Ophthalmological Society was organized in January, 1935, and its first meeting was held at Cheeloo University School of Medicine, Shantung, China, on January 30. Dr. Eugene Chan, a former member of the staff of the Wilmer Ophthalmological Institute of Baltimore and present head of the department of ophthalmology at Cheeloo, acted as chairman.

UNIVERSITY NEWS

Postgraduate Course in Ophthalmology and Otolaryngology.—The fifth annual postgraduate course in ophthalmology and otolaryngology will be offered by the Department of Post-Graduate Medicine and the Michigan State Medical Society at the University of Michigan from April 22 to 27. The first three days of the course will be devoted to otolaryngology and the last three to ophthalmology. The fee is $25, or $15 for either division. Enrolment is limited, and registration will be in the order of application. Checks, which are returnable if the applicant is unable to attend, should be mailed to the Department of Post-Graduate Medicine, University Hospital, Ann Arbor, Michigan.

Summer Courses in Sight-Saving.—Courses for the training of teachers and supervisors of sight-saving classes will be offered at the 1935 summer sessions of the following institutions on the dates indicated:

Western Reserve University, Cleveland, June 24 to August 2.

State Teachers College, Buffalo, July 1 to August 9.

Teachers College, Columbia University, New York, July 8 to August 16.

Information regarding the courses may be obtained from the institution offering the course or from the directors in charge, as follows: Olive S. Peck, Supervisor of Sight-Saving Classes of Northern Ohio, Board of Education, Cleveland.

Matie M. Carter, Supervisor of Sight-Saving Classes, State Education Department, Albany, N. Y.

Winifred Hathaway, Associate Director, National Society for the Prevention of Blindness, 50 West Fiftieth Street, New York.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Bacteriology and Serology

SEROLOGIC VALUE OF "TUBERCLE DE CARABELLI AND SABOURAUD" IN HEREDITARY OCULAR SYPHILIS. E. AUBINEAU, Ann. d'ocul. **171:** 671 (Aug.) 1934.

In syphilis, particularly in hereditary syphilis, it is the clinical examination which forms the primary guide of the practitioner. A positive Wassermann reaction is an argument in favor of syphilis, but this serologic reaction is so often negative in hereditary syphilis that too much dependence on it is dangerous not only for the patient but for his family and even for the physician.

The importance of syphilis in ocular pathology has been called to the attention of physicians by the communications of Sabouraud which appeared in the *Presse médicale* in 1917, by the thesis of Lefebvra in 1920, and by the articles of Hissard in 1930 and by Lamache in 1931.

The following clinical observations are detailed: Among 2,600 children between the ages of 8 and 13 years who presented themselves from 1924 to 1933, there were 91 carriers of "tubercles of Carabelli and Sabouraud." This percentage corresponded with that of Hissard (4 per cent). Among these 91 children presenting dental dystrophy there were found 73 with ocular involvements. The details of this observation are given in table form.

S. H. McKEE.

THE NOMENCLATURE IN ALLERGY: THE RELATION BETWEEN ANAPHYLAXIS AND IMMUNITY. W. RIEHM, Klin. Monatsbl. f. Augenh. **92:** 721 (June) 1934.

In the first chapter of his paper, Riehm offers valuable suggestions for clarifying the confusing terminology of allergy, and proposes new terms in place of those which render no exact definition. For example, anaphylaxis and immunity are demonstrated in connection with the presence of antibodies, yet an explanation is lacking for the conditions under which these antibodies cause anaphylaxis in one instance and immunity in another. Riehm suggests the use of the term "anaphylactic reaction" when the antigen-antibody reaction takes place in the cell of the body, and, on the other hand, the use of the term "immunity," meaning the destruction of pathologic noxa, when the reaction occurs in the microbic cell. The two processes do not exclude one another, but may occur simultaneously. Equally difficult is the determination of the terminology of the tolerance test, its mechanism and the quantity of material required for it. In determining the reaction of the organism toward each tolerance test, one must consider whether the irritating agent is living or dead, artificial or spontaneous, local or diffused, quick or slow of action. The forms of reaction are explained in detail, with the addition of explanatory examples, and illustrated by a table. The first rubric of this table comprises *Normergie* with normergic hyper-

sensitivity and hyposensitivity; the second rubric is *Heterergie,* with the subdivisions, *Tropergie* and *Immunergie,* the latter being subdivided into (1) *Metergie* with hyposensitivity to toxins and (2) *Allergie* with negative anaphylactogenous hyposensitivity, anaphylactogenous hypersensitivity and positive anaphylactogenous hyposensitivity. To the second rubric belongs, furthermore, *Immunergie* by means of lysins, agglutinins, precipitins, complement-binding antibodies and opsonins. Nonspecific deallergization with inhibition or increase of the production and function of the antibodies forms the third rubric.

Riehm formulates the relation between anaphylaxis and immunity as follows: Anaphylaxis is a lesion of the cell of the animal organism by antigens. Immunity is a lesion of the microbic cell by antibodies. Immunity is coupled with positive anergy if the action of the antibodies is high in comparison with the doses of infection; immunity is coupled with anaphylaxis if the relation is less favorable.

Dysimmunity with signs of anaphylactic reaction still prevails while conditions are getting more favorable for the noxa, until finally negative anergy develops in addition to total dysimmunity, presenting the most unfavorable omen.

K. L. STOLL.

Conjunctiva

UNIOCULAR CONJUNCTIVITIS FROM PEAT DUST. B. J. POWELL JR., Am. J. Ophth. **17:** 206 (March) 1934.

Powell describes the San Joaquin River delta and the irritating peat dust which it gives off during a hot, dry, windy season. He found a number of cases of conjunctivitis which originated in this area. The condition was characterized by profuse lacrimation, intense pain and photophobia, and it cleared up promptly under treatment with cocaine, homatropine and silver protein. The following summary is given: "This type of conjunctivitis is due to irritation from acid salts and silica spicules found in peat dust. It is mechanically aggravated by rubbing. A dry, windy climate is necessary. The essential point in treatment is to allay the spasm of the iris. It is always uniocular." W. S. REESE.

INJECTIONS OF BLOOD IN THE TREATMENT OF PHLYCTENULAR DISEASES IN CHILDREN. KUNITOMO, Acta soc. ophth. jap. **38:** 48 (June) 1934.

The mother's blood was injected intramuscularly into children with phlyctenular disease in which the symptoms of irritation were severe. At first 10 cc. was injected, and subsequently from 10 to 2 cc. was injected twice a week. The symptoms of irritation were much improved, and the progress of the disease was shortened without complications.

A. KNAPP.

Comparative Ophthalmology

OCULAR TROUBLES IN THE PARALYSIS OF FOWL. MORAX, TRUCHE and NIDA, Bull. Soc. d'opht. de Paris, February 1934, p. 83.

A hen sent to the Pasteur Institute showed no other symptoms except an inability to find her food. Examination of the eyes by slit lamp revealed a lesion of the irides characterized by depigmentation, synechia

and opacification of the lens. Microscopic examination demonstrated a chronic inflammation with infiltration by mononuclear and plasma cells in the region of the ciliary body. This symptom complex is thought to be an aberrance of the usual virus disease in which there is a paralysis of nerves, which usually affects the sciatic nerve. In the latter condition there are infiltration and hypertrophy of the nerve. The condition is comparable to certain chronic infections found in man, such as tuberculosis, syphilis and leprosy.

L. L. MAYER.

Cornea and Sclera

DYSTROPHY OF THE CORNEAL ENDOTHELIUM. E. L. GOAR, Am. J. Ophth. **17**: 215 (March) 1934.

Goar remarks on the paucity of literature on dystrophy of the corneal endothelium and enumerates the different titles under which it has been discussed. He gives a summary substantially as follows: 1. Dystrophy of the corneal endothelium or cornea guttata (Vogt) is a clinical entity occurring in some degree in about 6 per cent of persons above 20 years of age. 2. It usually begins in the fourth or fifth decade and is three times as common in women as in men. 3. It is a chronic, slowly progressive disease of the corneal endothelium, resulting in irregular thickenings and excrescences of Descemet's membrane. These excrescences are usually directed posteriorly and have staining properties identical with those of the membrane from which they are derived. 4. In advanced cases, this disease reduces the vision very materially, and this stage may be recognized by oblique illumination and by the ophthalmoscope. In early cases, the condition may be diagnosed only by biomicroscopy. 5. Epithelial dystrophy is often found in advanced cases, and it may be the late stage of the process which is represented by endothelial dystrophy at an earlier stage.

W. S. REESE.

BAND KERATITIS. R. J. SISSON, Am. J. Ophth. **17**: 222 (March) 1934.

Sisson gives a short history and reviews the literature of band keratitis, mentions the synonyms, and describes the two types, primary and secondary. He made a chemical study of flakes of the opacity and offers the following observations: The crystallographic examination indicated that this substance had an index of about 1.54 or 1.55, and was moderately doubly refracting, but that there was no crystalline outline. The impression was that silicon was present. In an advanced case of band keratitis of the secondary type, the x-ray spectra proved the presence of large amounts of calcium and phosphorus with small amounts of sulphur and silicon. It is possible that in the earlier stages of this condition the elements mentioned are present in such small amounts that they can be easily overlooked. The x-ray spectrographic method is suggested for the determination of the composition of the particles in the earlier cases. Treatment consists in "removal by scraping with a sharp spud." Under cocaine anesthesia, this is comparatively easy. "The large epithelial type may be removed almost intact. The subepithelial type may be chipped off. The substance is fairly firmly united to the

anterior layers of the substantia propria of the cornea. The cornea under the opacity is clear after the removal of the band. Spontaneous cures have been reported."

W. S. REESE.

A CASE OF CORNEAL CALCIFICATION (BAND-SHAPED KERATITIS) WITH CONJUNCTIVAL CHANGES. F. B. WALSH and E. CHAN, Am. J. Ophth. **17**: 238 (March) 1934.

Walsh and Chan report the case of a boy of 18 years who complained of defective vision in both eyes, because of which he left school at the age of 12 or 13. The patient's mother is married to her uncle, the patient being the only child of this union. At the age of 11, the boy complained of a sticking sensation in the right eye, and his mother noticed a white spot in the center of the cornea. Two years later a similar condition developed in the left eye. Physically the boy was poorly developed, poorly nourished and weighed only 94 pounds (42.6 Kg.). He was classified as feebleminded by a psychiatrist. The bulbar conjunctiva of the right eye was thickened and had a pearly glistening white color. It extended onto the cornea from above in the form of fingerlike processes. The conjunctiva immediately above the limbus had the dimpled appearance seen after some trephining operations. There was a dull, white, opaque band with irregular edges extending transversely across the cornea and stopping just short of the limbus. The iris was atrophic with anterior synechiae, and the tension of the eye was below normal. The left eye presented a similar condition, except that the corneal opacity was round and centrally placed. Below it was a white mass which almost filled the pupillary space. The authors discuss band-shaped keratitis and also the conjunctival changes.

W. S. REESE.

CRYSTALS OF CHOLESTEROL IN THE CENTER OF THE CORNEA WITH GERONTOXON. BONNET, PAUFIQUE and BONAMOUR, Bull. Soc. d'opht. de Paris, March 1934, p. 225.

A robust 31 year old man complained of pain, photophobia and tearing. A white speck was noted in each cornea. The epithelium of the cornea was normal, flat and translucent, and reacted well. In addition, on either lateral side, near the limbus, was a gerontoxon. The slit lamp demonstrated that the spot on the cornea was a fine glistening needle point. This was excised from each cornea with a von Graefe knife. In polarized light the needle points showed a double refraction, which disappeared after the application of ether. Therefore they were thought to be cholesterol.

L. L. MAYER.

KERATITIS NUMMULARIS DIMMER. M. SALZMANN, Arch. f. Ophth. **132**: 399 (Aug.) 1934.

The retired head of the Eye Clinic of the University of Gratz, Austria, in this paper reports his extensive experiences (eighty-eight cases) with that corneal disease which Dimmer first described in 1905 and which was recently brought into the foreground again by Aust.

The disease is most common in people between the ages of 15 and 45. Fresh cases are seen in largest number in September and October. Keratitis nummularis seems to be an occupational disease of the farmer. The condition is nearly always confined to one eye (only two bilateral cases against eighty-six unilateral cases). As to the initial symptoms, Salzmann believes that they are much like those of superficial punctate keratitis (Fuchs). From the beginning there are several lesions, accompanied by a "conjunctivitis" without secretion but with ciliary injection. The diagnosis of conjunctivitis, as made apparently in some early cases of keratitis nummularis, is inadequate, according to Salzmann, because ciliary injection always prevails. The typical corneal lesions have a diameter of from 1 to 3 mm. Through confluence, much larger and very irregular forms develop. The early appearance of a depression of the surface over the lesion without actual exulceration is very characteristic of keratitis nummularis. Salzmann, however, believes that this also occurs in superficial punctate keratitis. Actual ulcers occasionally develop in typical keratitis nummularis. Vascularization, superficial as well as deep, is not infrequent. The disease takes a sluggish course. Slight irritation and increased sensitivity of the affected eye persist for from nine to ten months. The complete resorption of the infiltrates and the formation of scars sometimes require two years. The uvea takes hardly any part in this type of keratitis. Follow-up work on patients with healed keratitis nummularis reveals the interesting fact that even after the scar stage has been reached some changes go on for years. The facets may grow, the outlines of the scars become fainter, and some lesions disappear completely. Change in the size of the facets in the pupillary area may for years continue to alter the refraction of the eye. The final scars can be studied more easily than the infiltrates. They are decidedly of two types, superficial and parenchymatous. The prognosis as to final vision is very good. Perforation of the cornea has not been observed by Salzmann. He knows of no specific treatment for keratitis nummularis. The favorable outcome in so many cases has made him very "lax" in the matter of treatment. He thinks keratitis nummularis is closely related to superficial punctate keratitis and to disciform keratitis. Keratitis nummularis occurs as a characteristic clinical picture which occasionally develops during the course of other well known corneal diseases (Salzmann quotes one case of dendritic keratitis) and as an occupational disease of farmers.

P. C. KRONFELD.

Experimental Pathology

PASSAGE OF ALCOHOL INTO THE EYE AFTER AN INTRAVENOUS INJECTION. L. MAZZI, Arch. di ottal. **41:** 21 (April-May) 1934.

A series of rabbits was given injections of alcohol in a dosage of 2 cc. of a 33 per cent solution per kilogram. At various times after the injections, both eyes of a rabbit were enucleated, and the amount of alcohol in the whole globe was estimated by distillation and by fractional determinations according to the method of Nicloux. Three minutes after injection, 3.25 mg. was present in the eye. The amount increased to 3.96 mg. after fifteen minutes, remained constant for one hour and then decreased until after eight hours no alcohol could be detected. When

maximal doses were given (7 cc. of a 33 per cent solution per kilogram) the amount of alcohol in the globe reached 3.75 parts per thousand.

In another series, the amounts present in the vitreous and lens were estimated. These were one-third larger than those found by Nicloux and Redslob after feeding alcohol by mouth, indicating the greater efficacy of the intravenous route. The proportions in the vitreous and lens were found to be the same by the two methods. In other animals the amounts in the vitreous and lens were compared with those in the blood at various times. It was found that a concentration approximating that in the blood was reached in the vitreous within one hour, while after three hours the concentration in the vitreous was higher than that in the blood. Diffusion into the lens was slower, the concentration never reaching six tenths of that in the blood during the time of observation.

An attempt is being made to observe the effect of intravenous injections of alcohol in cases of intra-ocular infection. S. R. GIFFORD.

THE PARATHYROID GLANDS AND OCULAR TENSION. G. ROSSI, Arch. di ottal. **41:** 195 (April-May) 1934.

In dogs, the parathyroid glands were extirpated together with half of the thyroid gland. The ocular tension was recorded daily with the Schiötz tonometer for from two to three weeks or until the death of the animal. In eight dogs the syndrome of cachexia parathyreopriva was produced. These animals showed a definite lowering of intra-ocular tension, which reached from 9 to 14 mm. in most cases. In one animal with tetany and a calcium content of 8 mg. per hundred cubic centimeters of blood, the use of an extract of parathyroid gland apparently caused a return of tension from 10 to 14 mm. No relation was observed, in general, between the lowering of calcium in the blood and that of intra-ocular tension. Symptoms of thyroid deficiency were not observed and Rossi is convinced that absence of the parathyroid glands was responsible for the symptoms observed. S. R. GIFFORD.

EXPERIMENTALLY PRODUCED HETEROCHROMIA. A. PASSOW, Arch. f. Augenh. **108:** 137, 1934.

Passow produced heterochromia iridis in rabbits by damaging the sympathetic nerve on one side. This could be accomplished only in young animals, before the end of the period of growth and development of the choroid. A lengthy dissertation follows on the differentiation of various forms of heterochromia which does not lend itself to abstracting.

F. H. ADLER.

INOCULATION OF CHICKENS AND MONKEYS WITH MATERIAL FROM HUMAN SYMPATHETIC OPHTHALMIA; REMARKS REGARDING THE NATURE OF THE VIRUS. A. VON SZILY, Klin. Monatsbl. f. Augenh. **93:** 145 (Aug.) 1934.

Von Szily refers to his research on the virus of corneal herpes with endogenous transmission of the infection from one eye to another. In

a similar manner the virus from an eye with sympathetic ophthalmia was inoculated into the eyes of chickens and one monkey. The uvea of the primarily inflamed eye, operated on for cataract four weeks prior to enucleation, was ground up with saline solution and injected into the vitreous. The inflammation could be transmitted from the human eye to the eyes of chickens and from these to the eyes of a monkey and those of other chickens. Like the herpes virus, the virus of sympathetic ophthalmia affected the contents of the orbit, the chiasm and the optic tract bilaterally. The effect differed, however, in three ways: The virus of sympathetic ophthalmia was not pathogenic to the cornea; it did not spread diffusely in the central nervous system, nor did it cause encephalitis or any general symptoms; on the other hand, it produced typical round cell accumulations, some of them resembling typical lymph follicles, which have been observed hitherto only in the conjunctiva. Their occurrence also in the uvea, orbit and optic tract seems to suggest an origin of the virus of ophthalmia in the conjunctiva. Formation of epithelioid cells and giant cells has been noted especially after transmission of the virus through a number of animals. Uniform lymphoid swelling of the uvea, interspersed with nests of epithelioid cells and giant cells, found in certain types of sympathetic ophthalmia in man, were absent in these animals. Von Szily introduced living tubercle bacilli into the blood stream of some of his animals, producing infiltration and typical tubercles. The reaction was less fulminant in the second eye. An inflammation akin to human ophthalmia could not be produced, nor did an intentional lesion of the ciliary body have any influence on the development of sympathetic symptoms. Sympathetic ophthalmia seems not to occur in animals; this may explain why its transmission to animals is so difficult. A large number of drawings and photomicrographs illustrate this paper.

K. L. STOLL.

General

FINDINGS OF EYE EXAMINATIONS: AN ANALYSIS OF 10,000 CASES. N. B. HARMAN, Brit. M. J., supp. 1562, Oct. 6, 1934, p. 181.

At a recent meeting of the British Medical Association, Harman as chairman of the ophthalmic committee, in presenting his report gave figures on the findings in a number of patients whose eyes were examined for glasses through the medium of the National Ophthalmic Treatment Board. These patients were referred to ophthalmic medical practitioners in different parts of the country.

Records of 10,000 patients were received from 47 doctors, and the following tabulation was obtained: Sixty-four per cent had errors of refraction only; 29 per cent had errors of refraction plus one or more other ocular conditions; 6 per cent had no error of refraction, but had one or more other ocular conditions; 1 per cent had no ocular defect.

The conditions other than errors of refraction were principally: cataract, 8 per cent; diseases of the conjunctiva and eyelids, 7 per cent; diseases of the uvea, 5 per cent; squint, 6 per cent; constitutional or ocular diseases, 5 per cent.

The main findings were that 1 in every 3 patients had some defect of their bodies or their eyes other than errors of refraction. This shows that tiring of the eyes and inability to see easily or clearly mean to most

people a need of glasses, which is natural enough, but the assumption may be right or it may be wrong.

The frequency of ocular conditions other than errors of refraction is plain evidence of the need of expert examination. It is particularly important to note the number of patients who do not require glasses.

There are many persons who believe that an examination of a patient's eyes by sight-testing opticians is both satisfactory and economical. A comparison of these returns with certain other returns shows that neither of these beliefs is borne out by facts.

Harman has attempted to find out how many patients were referred to ophthalmic surgeons by sight-testing opticians for further examination. This was a difficult matter to determine. He has, however, obtained figures from certain approved societies, which show that the percentage of patients referred by sight-testing opticians to ophthalmic surgeons ranges between 1 and 4.6 per cent.

In other words, in a series of patients examined by ophthalmic surgeons, 29 per cent proved to have conditions other than errors of refraction, and no fewer than 6 per cent had other conditions without any error of refraction, yet the percentage of patients referred for further examination by the opticians, who profess to be able to deal only with errors of refraction, was but 3 per cent.

This can have only one interpretation, namely, that opticians do not recognize defects in the eyes. Harman believes that these findings show an examination by an optician to be uneconomical, and, conversely, that approved societies or other agencies responsible for advising patients to seek ophthalmologic examinations cannot, in the interests of these patients, do other than adopt the only economical method, that of securing examination by competent ophthalmic medical practitioners.

A. KNAPP.

AUTOMOBILE ACCIDENTS AND HEMERALOPIA. J. ROLLET, Bull. Soc. d'opht. de Paris, January 1934, p. 48.

Rollet reports two accidents involving bus chauffeurs whose history of night blindness seemed to be the important factor. The fundi of these drivers were perfectly normal, but dark adaptation of their eyes was markedly retarded. Rollet suggests that the increase in accidents at night as compared with those in the daytime may be accounted for by such difficulties. Ability to become adapted should be one of the objects of examination in bus drivers and also in airplane pilots.

L. L. MAYER.

General Diseases

OCULAR PEMPHIGUS. B. W. RYCROFT, Brit. J. Ophth. **18:** 571 (Oct.) 1934.

Rycroft reports five cases of ocular pemphigus and discusses the relation of this condition to pemphigus of the skin. The relationship to syphilis has been noted, and a positive Wassermann reaction is frequently coincident with ocular pemphigus. The weight of evidence is against any bacterial cause. The differential diagnosis is discussed.

In the treatment of ocular pemphigus almost every remedy in fashion has been employed, but the results have been consistently bad. One patient was benefited by treatment. Whether the improvement was due to reduction of vascularity and redundancy of the mucosa or to true cure is a question.

The article is illustrated, and an extended bibliography is appended.

W. ZENTMAYER.

OCULAR SYMPTOMS IN PULMONARY TUBERCULOSIS. C. GIANNANTONI and G. POSSENTE, Ann. di ottal. **61:** 823 (Nov.) 1933.

One hundred and fifty patients with clinical and roentgenologic signs of pulmonary tuberculosis were examined with especial regard to anomalies of the pupils and the reaction of the pupils to atropine. Herman's test consists in the use of 0.1 per cent atropine, after which patients with apical lesions may show anisocoria because of involvement of the sympathetic chain. Sergent's test depends on the return of the pupils to normal size after the use of 2 per cent atropine. This occurs in from five to six days in normal persons, but requires from nine to ten days on the side of a pulmonary lesion.

The length of the cilia was greater than normal in one hundred and eight of the cases. Styes, chalazia and blepharitis were frequent among the younger patients. The pupillary tests revealed anisocoria in 64 per cent of the cases, the larger pupil being on the side of the pulmonary lesion in unilateral cases, or on the side most extensively involved in bilateral cases. Phlyctenular conjunctivitis was present in 22.66 per cent and follicular hypertrophy in 30 per cent of the cases. Heterochromia iridis was present in 17.33 per cent of the cases, the lighter iris being on the side of the most extensive pulmonary lesion. Anisocoria was most frequent in the more advanced cases of tuberculosis (65 per cent). It is interpreted as due to irritation of the sympathetic fibers in the thorax. The possible value of such observations in patients suspected of having tuberculosis is emphasized.

S. R. GIFFORD.

Glaucoma

TWO CASES OF BUPHTHALMUS IN SIBLINGS. C. HYMES, Am. J. Ophth. **17:** 132 (Feb.) 1934.

Two cases of buphthalmos occurring in sisters are reported. In the first the eyes were apparently normal up to the age of 6 months, at which time the parents observed that they were becoming enlarged. The right eye was operated on unsuccessfully and was phthisical. The left eye showed a steamy cornea, 17 mm. in diameter; the anterior chamber was deep and to palpation the tension appeared elevated. An Elliot trephining was done on this eye, following which the fundus could be seen, the optic nerve showing a deep glaucomatous cup. In the second case the parents noted that at the age of 7 months each cornea became hazy and the eyes were enlarging as had happened in the older sister. Examination revealed enlarged eyeballs and corneas, the latter being steamy. Tension to palpation was increased. A trephining was done on both eyes, following which the corneas cleared. About six years after

operation the first patient had vision of 5/200 in the left eye, and the second vision of 5/10 in the right and 5/5 in the left eye. Hymes feels that patients showing buphthalmos should be operated on early.

W. S. REESE.

THE RELATION BETWEEN GLAUCOMA AND DEFECTS OF HEARING. C. CORDERO and C. F. PORTA, Arch. di ottal. **41:** 159 (April-May) 1934.

A number of authors have noted an association of glaucoma with hearing defects and considered that the same factors may be responsible for both conditions. In the present study thirty-nine patients with primary and five with secondary glaucoma were given complete examinations, including tests of hearing with the audiometer. Of those with primary glaucoma, 30.75 per cent showed defects of hearing in which involvement of the nerve was the principal factor; 38.4 per cent showed defects due to tubal catarrh and middle ear conditions. Only 30.75 per cent had normal hearing. Of the five patients with secondary glaucoma, two had defects of the latter typé, and none showed nerve deafness. There was no relation between the severity of the glaucoma and the amount of the hearing defect. It is concluded that both the auditory and the visual apparatus are commonly affected by a primary vascular disease, a so-called "dysergia," which affects certain persons showing other vasodegenerative signs. A bibliography is appended.

S. R. GIFFORD.

THE RÔLE OF THE IRIS AND THE MOVEMENT OF THE PUPIL IN THE GENESIS OF GLAUCOMA: REPORT OF CASES. J. UUDELT, Klin. Monatsbl. f. Augenh. **94:** 619 (May) 1934.

Uudelt refers to two theses which he established four years ago as a result of his observations with the slit lamp: 1. Besides its function of warding off the light, the iris possesses also the power to regulate change of the aqueous humor by means of the action of the pupil, the crypts and the lymph spaces. 2. Senile degeneration of the iris due to atrophy or proliferation of connective tissue (and evident by disappearance of the pigment seam of the pupil, dispersion of the pigment, flattening of the trabeculae and consolidation of the iris tissue, especially in glaucoma) may be a cause of intra-ocular hypertension, as a result of obliteration of the lymph spaces. Uudelt discusses the reasons for his view and compares it with that of other authors. He refers to the occurrence of intra-ocular hypertension without obliteration of the angle of the anterior chamber, and to that of normal tension when this angle has been abolished. In explanation he points to the extraordinary development in the iris of spongy tissue, crypts, lagunas, blood vessels and lymph spaces. This structure and features of evolution prove the existence of a function of the iris which regulates the flow and formation of the aqueous humor.

To illustrate the occurrence of glaucoma as a result of primary atrophy of the iris, Uudelt cites a case reported by E. Fuchs and one observed by himself. In both cases anterior synechiae existed. In this connection, glaucoma in congenital and acquired aniridia is mentioned to prove that the age of the patient predisposes to the development of

glaucoma. Thus, atrophy and consolidation of the iris may be observed with the slit lamp in the nonglaucomatous eye of a patient with glaucoma in the other eye. Four cases of this type are reported. Tonometric examinations of glaucomatous eyes showing undulations of the intraocular pressure at different hours of the day, independent of the blood pressure, support Uudelt's contention. Furthermore, experiments on glaucomatous eyes kept in bright light and in the dark, respectively, showed that the hypertension decreased and the cornea often cleared in bright light, while a glaucomatous attack developed in an apparently nonglaucomatous eye which was bandaged after the operation on its mate. It was not the psychical effect of the bandage which produced this attack, but the forced rest of the pupillary movement, which stopped the action of the iris as a suction pump. Uudelt concludes: "It cannot be imagined that nature should have created a wonderful and complicated organ such as the iris, with its genetically heterogenous layers, for no purpose but to ward off the light."

K. L. STOLL.

POSTERIOR SCLERECTOMY IN ABSOLUTE GLAUCOMA. L. K. SOLOWIEFF, Sovet. vestnik oftal. **5:** 21, 1934.

Solowieff performed posterior sclerectomy on sixty-four eyes with painful absolute glaucoma. The simple technic of the operation is described. Complete relief from pain was obtained in twenty-eight patients. The operation had to be repeated in twenty-three patients. In fifty-one eyes, therefore, an enucleation and the resulting deformity were prevented. In thirteen cases no favorable results were obtained. In two of these a sarcoma of the choroid was found on enucleation. The advantage of the posterior sclerectomy over Elliot's trephine operation lies in the fact that there is no irritation of the ciliary body, the operative field being quite remote from the latter. Solowieff explains the mechanism of the operation as follows: When the tension increases to the degree that it produces severe pain because of pressure on the ciliary nerves, part of the lymph is forced out through the trephine opening, which unites the subchoroidal space with the subconjunctival cavity.

Solowieff believes that posterior sclerectomy should be tried in every case of absolute glaucoma whenever an enucleation is otherwise indicated, as it saves the patient from both physical deformity and psychic shock.

O. SITCHEVSKA.

PERMANENT ABSENCE OF THE ANTERIOR CHAMBER AFTER ELLIOT TREPHINING, ITS DEVELOPMENT AND PREVENTION. ICHIKAWA, Acta soc. ophth. jap. **38:** 46 (June) 1934.

One of the complications of trephining is a persistent abolishment of the anterior chamber. As this complication is not usual after other operations on the eye, the belief seems justified that the fistulization with possibly an excessive filtration of the aqueous is closely connected with its production. However, this cannot be the only cause, for the eye does not remain permanently hypotonic; frequently increased tension

results. The excessive filtration of the aqueous may retard restoration but cannot be responsible for permanent abolishment of the anterior chamber.

Ichikawa has recently performed an operation for cataract in a case of this kind, and made the unusual observation that there was an extensive adhesion between the cornea and the iris. This presumably resulted through hyperemia of the iris, following operative irritation. To prevent this complication, the energetic use of atropine after the operation is essential.

A. KNAPP.

Lens

THREE GENERATIONS OF GLASSMAKERS WITH CATARACT. J. SÉDAN, Bull. Soc. d'opht. de Paris, February 1934, p. 91.

Sedan operated on a man, 33 years of age, for cataract. He had been working for sixteen years in a glass factory. His father was operated on at the age of 25 after eight years employment in the same industry. His grandfather had a similar condition after fifteen years' work. The question of the condition being hereditary was eliminated by examination and inquiry into the history of members of the family not doing this work.

L. L. MAYER.

DISTURBANCE OF THE OXIDATION-REDUCTION OF THE CATARACTOUS LENS. J. NORDMANN, Bull. Soc. d'opht. de Paris, March 1934, p. 202.

Two grams of lens substance of the cow, dog, rabbit and man of different ages was mixed with 10 cc. of phosphate buffer ($p_H = 7$). In the electrode of Vles and Vellinger modified by Vellinger, the potential was determined in oxygenated nitrogen. As a reducing agent, sodium hyposulphite was used, and as an oxidizing agent, potassium ferrocyanide. The best results were obtained by oxidation. By this method there was found an s-shaped curve with a plateau, which for the most part was recorded between +150 and +130 millivolts (on the normal hydrogen electrode). The disturbance had the same duration in the lenses of old and young persons.

L. L. MAYER.

APROPOS OF A CERTAIN PHENOMENON OF OXIDATION-REDUCTION OF THE LENS. J. NORDMANN and P. REISS, Bull. Soc. d'opht. de Paris, March 1934, p. 203.

The measured oxidation potential of the lens falls in duration in vacuum or in nitrogen in four hours from +90 to —100 millivolts. The cataractous lens was observed by Goldsmith either to present no reduction of methylthionine hydrochloride or a reduction that was very late. The authors believe that because the potential is raised in a cataractous lens this phenomenon takes place. They cite their experiments with the lens in vitro to confirm this theory and are studying the lens in vivo.

L. L. MAYER.

EXPULSIVE HEMORRHAGE AFTER OPERATION FOR CATARACT. A. CUSUMANO, Arch. di ottal. **40:** 129 (March) 1933.

A case is described in which hemorrhage occurred ten hours after an uneventful extraction, preliminary iridectomy having been performed a month previously without mishap. On account of the general symptoms, vomiting and malaise apparently preceding the hemorrhage the author is led to theorize concerning a possible disturbance of the sympathetic nervous system as at least a contributing cause.

S. R. GIFFORD.

FURTHER INVESTIGATIONS OF THE TOXICITY OF THE LENS. REINHARD BRAUN, Arch. f. Augenh. **108:** 341, 1934.

The blood pressure was recorded from the common carotid artery of rabbits and cats under ether or a barbituric acid derivative anesthesia, and the changes due to injections of extracts of lens material were measured. The author concludes that aqueous and alcoholic extracts of fresh and dried lenses cause a reduction of the blood pressure. This fall in blood pressure is not due to either histamine or acetylcholine. Some unknown factor in the lens substance must be responsible. The inflammatory reactions of some eyes after operation for cataract are probably due to the toxic effect of lens material remaining in the anterior chamber.

F. H. ADLER.

THE ABSORPTION OF THE VISIBLE SPECTRUM BY THE HUMAN LENS. MAX BÜCKLERS, Arch. f. Augenh. **108:** 479, 1934.

Fresh human lenses were examined by the method of Spalteholtz, and the absorption of the visible spectrum by the lens was measured. The author finds that the human lens varies from a light yellow-green to the deepest reddish brown, and corresponding to this the absorption covers the region of extreme violet up to the orange. From this it follows not only that there is a blue blindness and relative yellow blindness caused by changes in the dioptric system of the eye, but also that there must be frequent violet blindness. The yellow color of the lens affords no protection against the short-waved light, but enhances distant vision.

F. H. ADLER.

THE EFFECT OF FRACTIONAL DOSAGE OF X-RAYS ON THE RABBIT EYE. H. GASTEIGER AND S. GRAUER, Arch. f. Augenh. **108:** 498, 1934.

A fractional dosage of x-rays caused falling out of the cilia, conjunctivitis and cataract in rabbit eyes. Histologically, damage to the corneal epithelium and to the retinal ganglion cells could also be demonstrated. Irradiation according to this method must be considered dangerous.

In concluding, the authors report a case of orbital tumor which was irradiated by this method. At the end of five months the eye was free from any clinical disturbance.

F. H. ADLER.

FURTHER INVESTIGATIONS ON THE HYDROPHILIC PROPERTIES OF THE LENS. F. P. FISCHER, Arch. f. Augenh. **108:** 517, 1934.

Fischer has continued his investigations on the binding of water by the lens reported in the *Archiv für Augenheilkunde* (**103:** 1, 1930; **108:** 80, 1933). In this paper he studies the intensity of this binding of water and finds that the lens has greater power of absorbing water than the outer coats of the eye. The lens swells in solutions of a strength, and after periods of exposure, at which the outer coats of the eye lose water, and with a much greater rapidity. Correspondingly, the lens maintains its water content to a much greater degree. While the outer coats of the eye reach a constant weight after an exposure to 37 C. for eighty minutes, the loss of water from the lens continues up to three hundred and thirty-six hours. For the most part, the water in the lens is bound to colloids, while in the external ocular coats only a very small portion is in this condition. This is of considerable importance in maintaining the transparency of the lens.

Further experiments show that the lens prevents, to a marked degree, the entrance of hydrogen ions and the exit and the loss of potassium, which cannot be satisfactorily explained on a simple, physical basis. Since the potassium content is always markedly lower in cataract, one must assume that in cataract formation some process interferes with this normal function of the lens, i. e., the maintenance of a constant potassium content.

F. H. ADLER.

THE FLUORESCENT SUBSTANCES OF THE LENS. F. P. FISCHER, Arch. f. Augenh. **108:** 544, 1934.

The conclusions reached by Fischer follow:

The deproteinated filtrate of a lens fluoresces the same as the lens does in vivo, and this persists even after treatment of the filtrate with Folin's silver lactate solution. A brownish-yellow mass can be obtained which has the same fluorescence as the lens and which is found more abundantly in the lenses of older animals than in younger lenses. In cataractous lenses this material is abundant. Its chemical nature is not clear, but it has not the characteristics of protein or carbohydrate or those of fat, and does not belong to the melanins or lipochromes. It has no reducing properties, contains no nitrogen or halogens, but does contain carbon, hydrogen, oxygen and sulphur, considerable potassium, traces of phosphorus and some iron. It is easily soluble in water, chloroform and ammonia.

After treatment of the filtrate with the silver lactate solution, vitamin B_2 is found in the precipitate and can be recovered by precipitation with 0.25 per cent hydrochloric acid. This fluoresces the same as the lens. There is less present in older lenses, and it disappears from cataractous lenses. It forms a reduction system, which the brownish-yellow substance previously described does not. Since the proteins of the lens, and all other substances which the lens contains, do not fluoresce, the fluorescence of the lens must be caused by these two groups which have just been described, i. e., vitamin B_2 and the brownish-yellow material, the chemical composition of which is not clear and which may be a mixture of substances. Vitamin B_2 is diminished in

older lenses and is absent in cataractous lenses, whereas the brownish-yellow substance is increased in age and is found in large amounts in cataractous lenses.

F. H. ADLER.

NEW EXPERIMENTAL DATA ON THE PATHOGENESIS OF CATARACT: PRELIMINARY REPORT. N. MONIUKOVA and M. FRADKIN, Sovet. vestnik oftal. **5**: 97, 1934.

This article presents an extensive physiologic chemical experiment on the rôle played by vitamin C in the production of cataract. Numerous chemical formulas illustrate the article.

By using Tillmans' reaction, Moniukova and Fradkin discovered a concentration of from 8 to 20 mg. of vitamin C, or ascorbinic acid (the empiric formula of which is $C_6H_8O_6$), per hundred cubic centimeters of aqueous humor in the anterior chamber of rabbits and men, while in the anterior chamber of cataractous patients hardly any ascorbinic acid was observed. They believe that ascorbinic acid plays an important rôle in oxidation and reduction of the lens. They made a careful experimental study of scorbutic animals, i. e., animals with avitaminosis C, in order to verify the recent data on the metabolism of both normal and pathologic lenses. In the first part of the experiment, twenty-five guinea-pigs were fed a scorbutogenous diet, hay and oats, for from fifteen to twenty days. The lenses were examined with the slit lamp, and in six animals incipient opacities were observed in the center and in the posterior layers of the lens in the shape of rings and punctate opacities. The second part of the experiment consisted of a careful puncturing of the anterior chamber with the tip of a keratome in eleven scorbutic guinea-pigs with transparent lenses. On examination with the slit lamp on the second or third day all these animals showed various-shaped opacities in the posterior layers of the lens. In the third part of the experiment, the same operation was done on a few animals which were getting food rich in vitamin C. None of these animals showed changes of the lens.

The next step of the experiment was to establish the correlation between vitamin C and the function of the endocrine gland system in cataractous patients, also the correlation between proteins and carbohydrates in the blood serum of these patients because of the presumption that high content of proteins and insufficiency of carbohydrates predispose to avitaminosis C. The Luthge-Metz reaction showed a disturbance of the function of the suprarenal gland in cataractous patients. There was also observed the absence of the cysteine reaction in cataractous patients, which indicated a disturbance of oxidation in the lens. The final step in the experiment was the feeding of the scorbutic animals which acquired cataracts with food rich in vitamin C. Iontophoresis and drops were used locally in the eyes. However, the time of observation of the last step was too short.

Moniukova and Fradkin come to the following conclusions: 1. Tillmans' reaction discovers in the normal anterior chamber of the rabbit a high concentration of vitamin C. 2. The aqueous of a cataractous eye has no ascorbinic acid. 3. The reducing property of vitamin C plays an important rôle in the oxidation processes of the lens. 4. The high concentration of vitamin C in a normal anterior chamber can be

explained by one-way diffusion of ascorbinic acid from the blood into the eye. 5. Opacity of the lens is observed only in severe, advanced cases of scurvy. 6. The emptying of the anterior chamber of the scorbutic animal leads to opacification of the lens. 7. A disappearance of vitamin C from the anterior chamber is an etiologic factor in opacification of the clear lens. 8. In the light of this experiment, senile cataract is a result of disturbance of the permeability of the eye on a background of general insufficiency of vitamin C.

O. SITCHEVSKA.

Neurology

ENOPHTHALMOS IN HORNER'S SYNDROME. H. P. WAGENER, Am. J. Ophth. **17**: 209 (March) 1934.

Wagener makes the following conclusions: "1. Demonstrable enophthalmos is a very inconstant sign of paralysis of the cervical sympathetic nerve. 2. Enophthalmos does not occur as an immediate result of cervicothoracic sympathetic ganglionectomy. 3. The enophthalmos associated with Horner's syndrome is therefore probably not due to relaxation of the smooth muscles of the lids and orbit. 4. A tendency to enophthalmos can be demonstrated in a series of cases of Horner's syndrome, although the enophthalmos is usually too slight to be definitely demonstrated by exophthalmometric measurements. 5. The enophthalmos is probably due to shrinking of the tissues in the orbit as the result of the loss of the trophic influence of the cervical sympathetic nerve. It is more likely to occur in cases of long standing, but is not seen in all such cases, even if the lesion is congenital. 6. Enophthalmos could be definitely demonstrated in only one of a series of ninety-four cases of clinical Horner's syndrome."

W. S. REESE.

MENINGOBLASTOMA OF THE LESSER WING OF THE SPHENOID. C. WESKAMP, Ann. d'ocul. **171**: 579 (July) 1934.

The meningoblastomas of Oberling, or the meningiomas of Cushing, were formerly grouped under the name of endothelioma of the dura mater because of the classic embryologic and histologic conception of the meninges. It is taught that the latter, proceeding from the mesoderm, is constituted of the pia mater, dura mater and arachnoid, representing a serous membrane with two surfaces, one visceral and the other parietal, covered with endothelium. It is natural, therefore, that tumors arising from the endothelium of the dura mater should be called endothelioma of the dura mater.

The details of the case of a man of 53 years are given. He came for consultation because of exophthalmos on the left. This had been present for some years. There were three important ocular symptoms: (1) exophthalmos, (2) alteration in vision and (3) subsequent alterations of the posterior pole of the orbital pyramid, demonstrated by x-ray picture.

He concludes that the exophthalmos, the alterations in vision and the characteristic alterations shown by the x-ray picture constituted a syndrome capable of establishing the diagnosis; and that this is probably the first report of a case of meningoblastoma of the lesser wing of the sphenoid published from the Argentine Republic.

S. H. MCKEE.

Operations

PRINCIPLES OF TECHNIC IN MUSCULAR ADVANCEMENT. H. LAGRANGE. Ann. d'ocul. **171:** 587 (July) 1934.

The only risk connected with the operation for strabismus has to do with placing the stitches properly so as to insure that the traction on the muscle will be correct.

The details of the technic are given under the following heads: I. Dissection: 1. Method of approaching the muscle body (*a*) a conjunctival buttonhole 2 mm. in length is made perpendicular to the muscle body; (*b*) the conjunctiva is carefully dissected to detach it from the sclera and capsule, and the dissection is continued until the back of the muscle is completely separated. 2. Dissection of the muscle body. II. Sutures: 1. Placing of the suture of correction. 2. Tension and reinsertion (with and without resection).

Lagrange believes the technic described is both simple and practical. Eight figures give good illustrations of the method followed.

S. H. McKEE.

THE CORNEA OF THE CADAVER AS MATERIAL FOR TRANSPLANTATION. V. P. FILATOFF, Ann. d'ocul. **171:** 721 (Sept.) 1934.

Since the first partial transplantation of the cornea with durable transparency, a report of which was published by Zirm in 1906, there have appeared in the literature reports of many cases in which the operation was successful. From 1922 to 1933 there were performed in the Ophthalmic Clinic of the Institute of Medicine of Odessa, Russia, ninety-six partial transplantations. This series resulted in permanent transparency in 14 cases, that is, in a transparent uniting of the transplanted material observed over a year (except in one case in which the observation had lasted for only one month and a half when the patient died).

Of the ninety-six transplantations, forty-eight were done on eyes with such complications as glaucoma, buphthalmos and leukoma. Transplantation of cornea on the eyes of patients with conditions of this type is never successful and generally should not be done. If one excludes these forty-eight cases, the fourteen cases in which the operation was successful give a percentage of 29.

In analyzing further the fourteen cases in which the transplantation was successful, Filatoff concludes that the reunion is most transparent when there is a large quantity of corneal element, confirming Elschnig's statement: "The best results are obtained when the transplanted material is surrounded with corneal element."

The details of two cases are given and there are eight illustrations.

S. H. McKEE.

OPTICOCILIARY RESECTION. M. J. AWERBACH, Sovet. vestnik oftal. **4:** 10, 1934.

Awerbach reviews the literature on this rather old operation. He estimates the value of the operation by an analysis of the results in 330 patients operated on in his clinic within a period of twenty years.

All suffered from absolute glaucoma with unbearable pain, which was not relieved by conservative treatment. The age varied from 18 to 83, but the majority of the blind patients were in the sixth and seventh decades.

The simple technic of the operation is described.

In 43 patients there were observed various complications: In 7 patients, a hemorrhage occurred. In 3 patients, marked exophthalmos developed, so that the eyes had to be enucleated. In 6 patients, the sclera was perforated, with prolapse of vitreous following. Ptosis was observed in 20 patients, or 6 per cent, and limitation of motion of the eyeball in 18 patients. In 12 patients there was muscle disturbance. There was a profuse hemorrhage during the operations in 4 patients, and in 2 a hematoma formed. The result of the opticociliary resection as regards the chief indication for the operation, the pain, was very effective, as in 308 patients the pain was relieved immediately after the operation. The intra-ocular tension remained unchanged. A venesection in patients with hypertension is considered to be a valuable measure for preventing bleeding.

Awerbach's conclusions are: 1. In all cases of severe pain, particularly in blind glaucomatous eyes, an opticociliary resection is the operation of choice instead of an enucleation. 2. Neurectomy as a rule stops the pain. 3. The appearance of the eyeball remains unchanged. 4. If the eyeball becomes atrophic, it can serve as a supporting stump for the prosthesis.

O. SITCHEVSKA.

Orbit, Eyeball and Accessory Sinuses

VARICOCELE OF THE ORBIT; EXTIRPATION OF THE VARICOSE MASS; LATE RESULTS. P. BONNET and L. PAUFIQUE, Bull. Soc. d'opht. de Paris, March 1934, p. 221.

A patient had been operated on two years previously for a varicocele of the base of the orbit. When the head was inclined forward, the volume of the orbit was augmented and the lower lid filled so that it looked like a roll. The operation consisted of dissecting out the venous channels and ligating them, then removing the varicocele in toto. One month after the operation, the clinical appearance was as if the tumor had recurred. However, this subsided, and now, after two years, the result is perfect.

L. L. MAYER.

Pharmacology

THE ACTION OF THE CHOLINES ON THE MUSCLES OF THE IRIS, WITH SPECIAL REFERENCE TO THEIR DOUBLE INNERVATION. K. VELHAGEN, Arch. f. Augenh. **108:** 126, 1934.

The effect of acetylcholine and carbaminocholine on the isolated iris shows that these drugs activate the sphincter even after a previous atropinization. The same concentration of the choline bodies suffices to stimulate both the dilator muscle and the atrophinized sphincter. Apparently there is a direct stimulation of the muscles of the iris by these drugs. These experiments lend support, according to the author, to

the theory of a double innervation of the sphincter muscle by sympathetic and parasympathetic fibers, but a single innervation of the dilator muscle.

F. H. ADLER.

N-METHYL-CYCLOHEXENYL-METHYL BARBITURIC ACID IN OPERATIONS ON THE EYE. H. KLITZSCH, Klin. Monatsbl. f. Augenh. **92:** 653 (May) 1934.

Klitzsch reports on experiences with a new general anesthetic, N-methyl-cyclohexenyl-methyl barbituric acid, at the University Eye Clinic of Halle, Germany. As the anesthetic is applied intravenously, the dosage has to be determined more or less individually. Young and vigorous persons require more than old ones; children, comparatively more than adults. A total of from 4 to 10 cc. of a 10 per cent solution of the drug is given by slow injection of doses of from 1 to 2 cc. until the patient slumbers without ceasing to react to mechanical irritation. Contrary to other published comments, no adverse results were met with by Klitzsch in one hundred cases.

This preparation is superior to ether because postoperative vomiting can be avoided, because the patient is wide awake directly after the operation, requiring no watching, and because postoperative excitement is absent. Total amnesia was present in each case. Preoperative examinations were made with special reference to the circulation of the blood and possible lesions of the parenchyma of the liver, as the anesthetic is split in the liver. The jaw is pulled forward during the operation so as to avoid asphyxia. The narcotic has been very successfully used in exenteration of the globe and of the orbit, in Krönlein's operation and in iridectomy for acute glaucoma. The latter operation was occasionally combined with venesection and letting of from 500 to 600 cc. of blood, and narcosis was obtained with 2 cc. or, at the highest, 4 cc. of the anesthetic. In the author's opinion, venesection reduces resistance to the anesthetic. Children tolerated two or three narcoses at intervals of from two to three weeks. Postoperative vomiting was not observed. The eyeball turned upward very rarely during the narcosis. The use of a local anesthetic and instillations of epinephrine hydrochloride are often combined with the administration of this narcotic.

K. L. STOLL.

EXPERIMENTAL INVESTIGATION OF THE INFLUENCE OF HISTAMINE ON THE EYE: FIRST COMMUNICATION. TAKABATAKE, Acta soc. ophth. jap. **38:** 50 (June) 1934.

The remedies used in glaucoma are related to the autonomic nervous system, and clinically the autonomic nervous system and glaucoma are associated. Histamine is an agent the action of which is much influenced by narcotics. The influence of histamine on the pupillary action is studied in this communication: 0.05 mg. of histamine injected intravenously causes a slight mydriasis; larger doses, shock and immediate death or coma with miosis; 10 mg. injected subcutaneously causes only slight mydriasis; 1.5 mg. injected subconjunctivally first produces miosis and then mydriasis; the stronger dose has the same effect; 0.2 mg. injected into the anterior chamber causes immediate miosis and

shock; instillation of a 10 per cent solution in the conjunctival sac causes miosis and then mydriasis. The reaction to light after the subconjunctival injection of histamine is always sluggish, both sets of muscles remaining rigid.

When histamine is applied locally there is a contraction and then a relaxation of the dilator and constrictor of the pupil which is retarded by narcotics, and the action of the autonomic nerves cannot be disregarded.

A. KNAPP.

Physiology

INFLUENCE OF VARIOUS ENDOCRINE PREPARATIONS ON THE SUGAR CONTENT OF THE OCULAR FLUIDS. NAKAYASU, Acta soc. ophth. jap. **38**: 49 (June) 1934.

Nakayasu has injected insulin, epinephrine hydrochloride, thyrotoxin and solution of pituitary gland subcutaneously or intravenously in order to determine the sugar content in the ocular fluids of rabbits, and has compared the results with determinations for the blood and the cerebrospinal fluid according to the Fujita-Iwatake method. He has also examined histologically the retina of rabbits' eyes for the amount of glycogen present during the stage of variation in the sugar content of the ocular fluids and has studied the relation between the sugar content of the blood and cerebrospinal fluid and the metabolism of glycogen in the retina.

He has made the following observations: From two and one-half to three hours after a single subcutaneous injection of a large dose of insulin there were signs of intoxication, with a diminution in the sugar content not only of the ocular fluids but also of the blood and the cerebrospinal fluid. The subcutaneous injection of a small dose of insulin produced a diminution of the sugar content not only of the aqueous of the anterior chamber, the vitreous and the cerebrospinal fluid, but also of the blood, together with a diminution of the retinal glycogen, with an increase in the hepatic glycogen. Repeated subcutaneous injections of 0.01 per cent solution of epinephrine hydrochloride led to an increase in the sugar content of the ocular fluids, blood and cerebrospinal fluid, which persisted for a number of hours. There was also an increase in the retinal glycogen and a slight increase in hepatic glycogen. Ten hours after an injection, the sugar content began to fall, and after twenty-four hours it returned to normal. Repeated injections of thyrotoxin caused no increase of sugar content, but the reduction in glycogen in the liver and in the retina was definite. After injection of a solution of pituitary gland there was a transitory increase of the blood sugar. The glycogen content of the liver was decreased, while there was little variation in that of the retina. It could not be definitely determined whether after the injection of a solution of pituitary gland there was an increase in retinal glycogen, or not.

A. KNAPP.

Refraction and Accommodation

THE CHANGES IN REFRACTION IN DIABETES. F. P. FISCHER, Arch. f. Augenh. **108**: 422, 1934.

In 1933, Granström (*Acta Ophth.* **11**: 1, 1933) suggested that the changes in refraction which often occur in diabetic patients are due to

changes in the nucleus of the crystalline lens, due to a retention of salt. Such a retention of salt in the lens leads to an increase in the refractive index of the eye as a whole and thus produces transitory myopia.

A similar retention of salt had been found by Klein in other tissues of the body, in that there occurs in the diabetic patient a change in the proportion of colloid-bound water to free water. In such cases the colloid-bound water is decreased, which accounts for the rapid loss of body weight. When a diabetic patient is given insulin, the power to bind water in colloid form is recovered, and the retention of salt ceases to be a "dry" salt retention and becomes a "wet" salt retention. According to Granström, the transitory myopia occurs during the stage of "dry" salt retention in untreated diabetic patients, whereas in the stage of "wet" salt retention during treatment, hyperopia may occur.

Fischer agrees with Granström and cites a case which he previously reported (*Arch. f. Augenh.* **103:** 55, 1930) which tends to support Granström's theory. He points out that such changes in the refraction in diabetic patients indicate a change in the permeability of the lens, and since a change in the permeability of the lens is probably the first stage in the development of cataracts, these changes in refraction should warn one of the danger of cataract formation, and the condition should therefore receive immediate attention and treatment. He believes that at this stage appropriate treatment of the diabetes can effectively check the process.

The author then points out that the permeability of the lens capsule may be quite different from that of the lens tissue itself, which he believes is quite independent of the former.

F. H. Adler.

Retina and Optic Nerve

Hereditary Optic Atrophy (Leber's Disease). A. J. Bedell, Am. J. Ophth. **17:** 195 (March) 1934.

Eight cases of Leber's disease occurring in five generations from three separate parent stocks are reported. These cases had been under observation since 1909. Syphilis was not a factor, and x-ray pictures of the sella turcica and optic foramen yielded no clue as to the cause of the disease. The following findings were common to all these cases: 1. The disk was sharply and clearly outlined. 2. There was a definite loss of substance, resulting in an atrophic cup. 3. The arteries were somewhat attenuated, but presented nothing characteristic either in their course or in their contour. 4. The loss of vision was sudden, usually within a few hours, and most often occurred over night, generally affecting both eyes, but sometimes to an unequal degree. Bedell concludes that "Leber's disease is essentially a familial atrophy of the optic nerve."

W. S. Reese.

A Case of Angiomatosis Retina. W. Niccol and R. F. Moore, Brit. J. Ophth. **18:** 454 (Aug.) 1934.

A man, aged 32, during an attack of "flu" noticed a black spot before the right eye and later had metamorphopsia. Vision was 6/36. The visual field showed enlargement of the blindspot toward the fixation point. The peripheral field was full.

Immediately adjoining and partly overlying the temporal edge of the papilla was a swelling of a peculiar irregular formation, seeming to be composed of a collection of small spherules clustered together, giving somewhat the appearance of a raspberry. Its general color was slate gray; some of the spherules had a reddish or purplish color and evidently contained blood.

Treatment by insertion of a radon seed was suggested, but as a third surgeon thought malignant growth could not be excluded, the eye was enucleated. The mass was composed of many irregular-shaped spaces of various sizes, lined by endothelial cells and containing blood. The outer nuclear layer of the retina was disorganized, and the rods and cones in the affected area were compressed and destroyed. Neuroglial tissue was pressed between cavernous spaces. There were areas of edema, hyaline degeneration and extravasated blood. The pathologic diagnosis was angiomatosis of the retina.

The article is illustrated.

W. ZENTMAYER.

DETACHMENT OF THE RETINA SECONDARY TO SARCOMA OF THE CHOROID. C. DÉJEAN and L. VARNIER, Arch. Soc. d. sc. méd. et biol. de Montpellier **15:** 144, 1934.

Of fourteen eyes enucleated for absolute glaucoma at the ophthalmic clinic in Montpellier, France, two revealed clinically unrecognized sarcoma of the choroid. In presenting a case in which the condition was diagnosed, the following points are emphasized: Most cases are seen after detachment has occurred. The detachment due to tumor is generally without folds, in an atypical location, and may show a double vascularization—retinal and tumoral. Transillumination is sometimes unreliable, as the light may be transmitted through a small leukosarcoma, or it may be blocked by intra-ocular hemorrhage.

A significant early subjective sign is the perception of a spot in the field of vision, particularly if yellowish. The mapping of this positive scotoma may locate the site of a tumor still ophthalmoscopically invisible. While the eye still maintains a normal visual acuity, a poor discrimination for green may be elicited in a part of the field, a progressive hypermetropia of from 1 to 4 diopters may develop, and in addition, there may be a localized edema of the retina and some increase in intra-ocular tension.

J. E. LEBENSOHN.

BEGINNING OF RETINAL DETACHMENT. R. BIDAULT, Bull. Soc. d'opht. de Paris, January 1934, p. 45.

Bidault reports his observations on a woman of 55 years whom he examined one year previous to the occurrence of a detachment of the retina. She had myopia of from 14 to 16 diopters in the left eye. There had never been any attempt to correct this error. The ophthalmoscope revealed a posterior staphyloma, a pale retina, marked delineation of the choroidal vessels, a lesion of the macula and white striae in places bounded by pigment lines. The vision was 1/30. The macula of the right eye was slightly discolored, and a zone of relatively pale retina surrounded the macula. The vision was 5/10. A year later vision was

entirely lost in the right eye. A yellowish discoloration was found in and surrounding the macular region. This degeneration of the macular region is thought by Bidault to be the cause of the retinal detachment.

L. L. MAYER.

FAMILIAL MACULAR DEGENERATION ASSOCIATED WITH RETINAL HEMORRHAGES. L. MAZZI, Arch. di ottal. **41**: 225 (April-May) 1934.

A man noted diminution of vision at the age of 44. Vision in the right eye was 4/10; in the left, 3/10. The maculae showed symmetrical areas of atrophy, surrounded by a ring of pigment. The peripheral fields were normal, but absolute scotomas were present at the fixation points. The periphery of the fundus and the retinal vessels were normal in each eye. The patient's son, a man of 25, had noted a marked reduction of vision in the left eye following a parachute descent three years before. A similar defect developed in the right eye, which improved after a time. At this time hemorrhages in the retina were noted by a reliable ophthalmologist. When the patient was seen by Mazzi vision in the right eye was 5/10; in the left, 10/10. The right eye showed a central area of atrophy much resembling that in the father, while the left eye, although no central field defect could be made out, showed a small black spot in the fovea surrounded by a white ring the size of the macular area. The condition is considered to fall into the group of progressive familial macular disease without cerebral degeneration described by Stargardt. Previously, normal vision was apparently present, since an aviator's examination was passed at the age of 20. The presence of hemorrhages is unusual, and a special lability of the retinal vessels must be assumed.

S. R. GIFFORD.

CONSTITUTIONAL CONDITIONING OF CERTAIN DISEASES OF THE MACULA LUTEA. R. SCHEERER, Klin. Monatsbl. f. Augenh. **92**: 467 (April) 1934.

More important than the clinical diagnosis is the recognition of constitutional diseases which necessarily lead to disorders in certain organs. Taking as an example the senile changes in the macula lutea, Scheerer discusses the question whether they are caused by wear or by increasing inefficiency of the vascular system. Furthermore, why do some persons suffer from Haab's degeneration of the macula and others from the edematohemorrhagic or fatty or cystoid forms? He succeeded in classing the foremost types of diseases of the macula as constitutional disorders of a vascular etiology.

The first group includes persons with a solid vascular system, who die from senile debility at a high age. Cessation of function of minute capillary areas of the body, even in the choroid and retina, does not cause great sudden disorder. The macula lutea, however, depending on an especially dense net of capillaries, suffers severely as soon as the blood supply sinks below a certain level. In cases of this type are found, ophthalmoscopically, so-called sclerosis of the choroid, changes in the pigment of the retina and occasionally minute retinal cysts in the posterior pole.

The second group includes vasoneurotic, pyknic or genuinely hypertonic patients with vividly changing activity of the capillaries. Their death is caused by severe peristatic hyperemia in the vital organs of the body. The same process in the retina produces macular disease, such as retinitis circinata, arteriosclerotic retinitis, hemorrhagic glaucoma and some forms of so-called albuminuric retinitis.

K. L. STOLL.

CYSTOID DEGENERATION OF THE RETINA AND ITS ACCOMPANYING NET-FORMING LINES; THE OPTIC CONDITIONS FOR THE VISIBILITY OF THE CYSTS. A. VOGT, Klin. Monatsbl. f. Augenh. **92:** 743 (June) 1934.

Cystoid degeneration of the periphery of the retina has been described by Vogt and other writers. Large cysts in the macula may be taken for formations of macular foramina. Examination by red-free light allows exact differential diagnosis in macular cases; in peripheral cases, diagnosis may be more difficult. Observation in the latter cases is easier in the detached retina: the cystoid design disappeared after operative reattachment and was observed again in cases in which the retina became detached later on. The cysts observed by Vogt were closed, with exception of one case in which a foramen existed; this is of therapeutic importance. White lines and meshes of white lines observed ophthalmoscopically in peripheral cystoid degeneration of the retina are obliterations of blood vessels. Vogt's article is illustrated by a number of pictures, in which the white lines are directly continued by blood vessels.

K. L. STOLL.

Trachoma

CONTRIBUTION TO THE QUESTION OF TRACHOMA OF THE TEAR SAC: REPORT OF CASES. F. VON PAPOLCZY, Klin. Monatsbl. f. Augenh. **92:** 613 (May) 1934.

The question whether trachoma occurs in the tear sac has not been answered. Follicles resembling those of trachoma have been observed in nontrachomatous conditions of the tear sac. Stock pointed out that ulceration of the epithelium covering the follicles and voiding of their contents into the tear sac are no proof for the presence of trachoma there. Papolczy examined histologically eight tear sacs of patients with trachoma of the conjunctiva and fourteen of patients suffering from ordinary inflammation of the tear sac. There was no histologic difference between the two types, which presented the same changes, consisting in diffused infiltration of the mucous and submucous layers, vascularization and hyaline degeneration. One tear sac with large follicles in great numbers was the exception; follicles were found in the tear sac in one half of the cases of trachoma. The problem can be solved only if trachomatous involvement by continuation in the canaliculi, as well as in the tear sac, can be demonstrated and inclusion corpuscles can be found.

K. L. STOLL.

Visual Tracts and Fields

MODIFICATIONS OF MARIOTTE'S BLINDSPOT UNDER THE INFLUENCE OF NASAL EXCITATION. A. MAGITOT and A. DUBOIS, Bull. soc. d'opht. de Paris, February 1934, p. 85.

Using Evan's method of angioscotometry, the authors compared the fields of normal persons taken before and after using a tampon saturated with a 10 per cent solution of cocaine hydrochloride in the nasal cavities. While the results varied somewhat in each patient, in the majority there was an enlargement of the blindspot following cocainization. The enlargement was bilateral and lasted about six minutes, after which there was a return to normal. The authors submit their results in order to show how vasomotor phenomena may influence blindspot determinations. Certain operative procedures on the sinuses when retrobulbar lesions are suspected often bring relief. The authors believe that this relief may be in part due to vasomotor changes.

L. L. MAYER.

STUDY OF THE VISUAL FIELD IN THE SYNDROME OF HYPOPHYSEAL HYPERFUNCTION. DROUET, JEANDELIZE and GAULT, Bull. Soc. d'opht. de Paris, March 1934, p. 205.

To the newer examinations of serum and urine for the biologic determination of hypophyseal disturbances, the authors have added a study of the visual fields. The conditions presented in eighteen patients were grouped as of four clinical types: exophthalmic goiter, eleven patients; syndrome para-exophthalmic goiter, four; arterial hypertension, two, and migraine, one. The indication for the studies of the visual fields was the finding of melanophores in the urine, denoting an increase in the function of the pituitary body. Other biologic tests included the reaction of Aschheim and Zondek, investigation of the thyroid stimulant of Max Aron, determination of prolan and the reaction of the erythrophores of Zondek.

The patients with hyperthyroidism were divided into a group of eight having treatment only and a group of three having thyroidectomy. In the former division were four patients with bitemporal recession of the fields, including one patient in whom only the red and green fields were constricted. Of the patients with surgical intervention, one was examined one month, another one year and the last two years after operation. Two showed retrenchment bitemporally, one for white alone, and the other for red and green.

The four patients with para-exophthalmic goiter all had the pituitary hormone in the urine, and three presented a constriction bitemporally, two for white, and one for red and green only. The clinical diagnosis was made in these cases by the signs of hyperthyroidism without increased metabolic rate.

In the hypertensive group the influence of the pituitary gland on the suprarenal glands was noted. Both patients showed melanophores in the urine and bitemporally contracted fields. The arterial hypertension was thought to be of endocrine origin. Treatment of the pituitary body with radium reduced the hypertension.

The one patient with migraine was seen at the time of attack and showed marked bitemporal constriction. The work of Riley, Brichler and Kuzok has demonstrated that elimination of the urinary prolan prevents migraine.

The authors believe that study of the visual fields is an important addition to the investigation and diagnosis of conditions of these types.

L. L. MAYER.

THE RELATION BETWEEN THE VISUAL CENTER AND THE VISUAL FIELD. ASHIKAGA, Acta soc. ophth. jap. **38:** 53 (June) 1934.

Ashikaga had the opportunity of examining a soldier, 23 years of age, who had been shot in the right occipital region. The x-ray picture showed a small foreign body situated 4.9 cm. from the back of the occiput, this placing it presumably in the anterior part of the calcarine fissure. The patient exhibited inferior homonymous hemianopia with preservation of the macula. On awakening, the patient was always blind for five or six seconds, and after from forty to fifty minutes the vision returned. Pressure on the right occipital region was followed by diminution of the vision from 0.7 to 0.4, and a central scotoma then appeared in the visual field. The eyegrounds remained normal.

Among the persons with head injuries observed during the Russo-Japanese war, ninety-two showed visual field disturbances: Five had a central scotoma, six a paracentral scotoma and fifty-six peripheral contraction. It is interesting to study the relation of these disturbances of the visual fields to injuries of the visual center.

The author has recently made some exact investigations, using a 3 to 5 mm. object at 40 cm. distance. He has found that there occur physiologically in healthy eyes about the point of fixation two concentric relative ring scotomas of oval form and of a breadth of from 2 to 3 mm. One lies from 8 to 9½ degrees and the other from 13 to 15 degrees from the point of fixation. There is also a relative blind line which runs vertically through the point of fixation.

The author thinks that these changes in the normal field should be of value in examining topical defects in the cortical visual center.

A. KNAPP.

Sympathetic Ophthalmia

THE PATHOGENESIS OF SYMPATHETIC OPHTHALMIA WITH REMARKS ON SYMPATHETIC RETROBULBAR NEURITIS. OGUCHI, Acta soc. ophth. jap. **38:** 98 (June) 1934.

In Elschnig's theory a second factor has always been missing, namely, the cause of the attack on the second eye. The theories that the sympathetic ophthalmia is a modified anaphylaxis have not been accepted, and Guillery's theory that it is tuberculotoxic has not been confirmed by Kiyosawa's experiments. Golowin was the first to suggest that it might be due to uveal cytotoxin, and Nakamura then published his theory that it represents an auto-immunization.

Oguchi reports the case of a farmer, 39 years of age, who had an extraction of cataract following an injury with a piece of wood. Two months later iritis developed in the other eye, accompanied by neuro-

retinitis. The eye involved first was removed, but the other eye did not improve. The enucleated eye showed a thickening of the choroid. The infiltrating cells were small lymphocytes accompanied by eosinophil leukocytes with Russell bodies. The sclera was infiltrated along the vessels and nerves as they proceed from the choroid, and in these regions numerous chromatophores and pigment with eosinophil leukocytes could be seen. Similar infiltrations occurred in the optic nerve, and very far back there was the picture of perineuritis and interstitial neuritis. It is striking that both chromatophores and pigment bodies could be traced very far back in the optic nerve by following the eosinophil cells.

Oguchi believes that the presence of these chromatophores is not accidental, but that they are a cause for the inflammation. The chromatophores liberated from the uvea act as antigen and as allergen. If the uvea is traumatized or the tumor is crushed, immune bodies occur in the tissues, which then attack and destroy the neighboring chromatophores, until this change takes place in the entire uvea. As all human tissues may contain these immune bodies, the liberated and wandering chromatophores produce an allergic infiltration when they are in a sufficient quantity, or the tissue is electively sensitized. The chromatophores themselves are destroyed just as the tubercle bacilli are in the allergic state of tuberculosis. This explains the infiltration of the sclera and of the optic nerve. If the liberated chromatophores pass on to the other eye, sympathetic ophthalmia develops.

The wandering of these cells may occur through the blood stream. This explains the attacking of the second eye if the uvea has previously become sensitized, and also explains such developments as deafness and the graying of the hair. The second path is the optic nerve; the third is the orbital facial venous system. The infiltration of the sclera can extend into the orbit, so that extirpation of the orbital tissue may improve the sympathetic ophthalmia. Nakamura thinks that autoimmune bodies may directly attack the organ of the other side. Oguchi believes, however, that it is necessary to have organ-specific albumin present, which acts as an allergen, in order to destroy the organ. This occurs in the liver as well as in the kidneys. Finally, the simultaneous appearance of anaphylaxis must be considered.

It has frequently been accepted anatomically that septic endophthalmitis can occur as the result of anaphylaxis, but one must remember that injection of the poison has been made into the vitreous. Sympathetic ophthalmia sometimes is associated with the endophthalmitis.

Eosinophil cells are frequently present in allergy. Specific uveitis with diseases of the hair has some similarity to sympathetic ophthalmia, and it may also be the result of liberated and wandering chromatophores which act as allergens, though their origin or any trauma is uncertain.

Oguchi adds a case of sympathetic retrobulbar neuritis. After exenteration of the eyeball, iritis and choroiditis appeared in the second eye without any changes in the macular region. There was a central scotoma. The course was favorable and the condition was cured.

A. KNAPP.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

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Oct. 15, 1934

GUERNSEY FREY, M.D., *Secretary*

WEBB W. WEEKS, M.D., *Chairman*

UNILATERAL EXOPHTHALMOS PRODUCED BY MENINGIOMA IN THE MIDDLE CRANIAL FOSSA: REPORT OF A CASE. DR. MARTIN COHEN.

This article will be published in full, with the discussion, in a later issue of the ARCHIVES.

A CASE OF FASCIOLA HEPATICA WITH OVA IN THE VITREOUS. DR. ISADORE GOLDSTEIN.

This article will be published in full, with the discussion, in a later issue of the ARCHIVES.

METASTASIS IN THE CHOROID FROM CARCINOMA OF THE TESTIS. DR. DAVID WEXLER.

This article will be published in full, with the discussion, in a later issue of the ARCHIVES.

Nov. 19, 1934

GUERNSEY FREY, M.D., *Secretary*

WEBB W. WEEKS, M.D., *Chairman*

SILVER OXIDE DEPOSITS IN SKIN, CONJUNCTIVAE AND CORNEAS. DR. JAMES W. SMITH.

On Dec. 31, 1931, a man aged 60 sustained burns of the face, hands and eyes as the result of an explosion of potassium oxalate and silver nitrate. The skin of the face, lids, ears, nose, lips and forehead was stained black by the deposition of silver oxide. The patient had experimented with many reducing or bleaching agents on a small area of skin on the left hand without clearing the discoloration. Prior to the accident, the vision in both eyes was normal.

The corneas were burned at the time of the explosion and showed central gray scars. In the periphery of the right cornea superotemporally and in the left cornea inferiorly were seen black grains of silver oxide. The visual acuity was 20/100 in the right eye and 20/100 in

the left eye. Local therapy failed to clear the corneal deposits or to improve the visual acuity. Optical iridectomy was considered, but no satisfactory area of clear cornea could be found.

DISCUSSION

DR. ERNST WALDSTEIN: About three or four years ago a paper appeared in the *Journal of the American Medical Association*, giving the results of treatment in several cases of argyrosis that resulted from the use of silver nitrate. An injection of sodium thiosulphate and either potassium ferricyanide or potassium ferrocyanide was used with very satisfactory results.

DR. VON ZELINSKI: I should like to suggest subconjunctival injections of a 1 per cent solution of sodium iodide. It has the power of combining with silver salts and is relatively harmless. Four or five minims (0.24 or 0.3 cc.) of a 1 per cent solution may be used for an injection.

A CASE OF SEVERE SYMPATHETIC OPHTHALMIA. DR. O. P. PERKINS.

Clinical History.—A 4 year old girl received an injury to the left cornea with a slight prolapse of the iris and trauma to the capsule of the lens. The corneal wound was freed from iris tissue by means of an iridectomy done through a keratome incision at the limbus. Three weeks later the eye showed only a moderate injection near the wound. An anterior adhesion of the capsule of the lens could be seen with the slit lamp. Also, on the anterior surface of the lens, in a localized area which corresponded to the operative coloboma, there was visible a small mass of tissue which was regarded as an organized vascular exudate. The iris tissue looked normal, there were no posterior corneal deposits, and the tension was reduced.

The right eye was normal in all respects until four weeks after the accident, when it suddenly showed a definite ciliary injection. The injured eye was enucleated within twenty-four hours after the appearance of this injection. Despite vigorous local and systemic treatment, the iridocyclitis in the sympathizing eye progressed unremittingly, and six months later the eye bordered on a state of phthisis and the patient was completely blind.

Pathologic Examination (Dr. Brittain F. Payne).—Examination of the specimen submitted by Drs. Knapp and Perkins revealed a ragged perforating wound on one side of the cornea and an operative wound at the limbus. The wounds were filled with an infiltration which extended back into the iris and ciliary body on the same side. The iris and ciliary body of the opposite side showed comparatively little infiltration. The outstanding feature was that practically all the foci were confined to the same side as the perforating wound.

The diagnoses were: sympathetic ophthalmia, perforating wound, traumatic iridocyclitis and traumatic cataract.

DISCUSSION

DR. ARNOLD KNAPP: The tragic feature of this case was that we were fully aware of the possibility of sympathetic ophthalmia and

watched the child every day most carefully, frequently with the slit lamp. We could not convince ourselves that the injured eye should come out; then, wholly without warning, the involvement of the second eye started up. This case confirmed the well known fact that the condition of the second eye runs an entirely independent course, and therefore the question naturally arises: What did we gain by removing the first eye? It is perfectly possible that if we had kept the first eye there might have been a prospect of some sight in that eye, but this is problematic.

DR. WENDELL L. HUGHES: I recall a similar case in which the removal of the primarily affected eye after involvement of the second eye did not prevent the disastrous course of sympathetic ophthalmia.

DR. MARK J. SCHOENBERG: I agree with Dr. Knapp that it is of no use to remove the first eye after the second eye has begun to be involved.

DR. MARTIN COHEN: I should like to ask if the vision became progressively worse from the start. The second question is whether owing to the marked toxemia the child showed evidence of hypotony very early, and whether one should attach more significance to varying intra-ocular pressure as indicative of severe iridocyclitis. I think that the tension was stated to have been reduced very early in the disease.

DR. JACQUES LANDESBERG: I recall a case of a perforating wound in the dangerous zone in which enucleation was not performed and sympathetic ophthalmia did not develop, and also a case of sympathetic ophthalmia following cataract extraction. In the latter case enucleation was also not performed, and some vision was preserved in the exciting eye whereas the second eye lost all perception of light.

DR. O. P. PERKINS: In answer to Dr. Cohen: There was undoubtedly a progressive loss of vision, but the progress in the cataract might have accounted for this. Just prior to the enucleation there was only perception of light. The tension was low from the beginning.

PRESENT METHODS OF OPERATING FOR DETACHMENT OF THE RETINA IN EUROPE. DR. ARNOLD KNAPP.

The ways in which the operation of electrocoagulation is being done abroad are described. In general, there is a tendency to return to surface coagulation in place of the deep penetration coagulation. The newer methods employing pyrometric electrodes of Coppez, Brussels, Meesman and Berlin are also described.

DISCUSSION

DR. MARK J. SCHOENBERG: Our European colleagues are apparently not entirely satisfied with their results, or they would not try modifications of the original methods of Larsson, Weve and Šafář. While electrosurgery is superior in many respects to the previous methods, there are still many unsolved problems. One of these problems is how much current to use to obtain the optimum result. Coppez and Meesman have devised their apparatus for standardizing the amount of heat developed in the choroid, yet even this attempt does not over-

come all the difficulties, since all sclerae are not equally resistant; on the same eyeball some areas are more resistant than other areas; some detachments are very shallow; others are ballooned; the subretinal fluid may be more or less gelatinous. All these and possibly other variations prevent a uniform result when using a standardized uniform temperature of 80 C.

Dr. Šafář is still using his original method, and I understand his results are quite satisfactory. His electrodes, however, are difficult to handle and to cleanse. For this reason I have devised a hook electrode which is made of platinum iridium and has a two-prong hook with very sharp points. It acts both as electrode and retractor, pulling the posterior portion of the eyeball forward and thus making it possible to reach as far back as necessary. It can be introduced into the sclera with ease and can be sterilized and cleansed by being passed through a Bunsen burner. The choroidal coagulations obtained by the use of this hook electrode are discrete and very small, and the results are satisfactory in suitable cases.

Dr. Arnold Knapp: I should have said that this electrode of Coppez also comes in a curved model so that it can be applied on areas situated farther back on the eye. The effect of Coppez' surface coagulations is only on the choroid; the sclera is not affected provided the temperature of 80 C. is used. With a temperature of 90 C., a slough of the sclera results. The purpose of the operation is to limit the effect to the choroid, and then, after successful evacuation of the subretinal fluid, the intact retina comes to lie on the irritated and coagulated choroid. A difficulty may be encountered in such cases as those to which Dr. Schoenberg has referred, in which there is a gelatinous exudate, or in which for some reason or other the subretinal effusion cannot be emptied, or in which the detachment happens to be in the upper part of the eye, where gravity does not at once replace the retina in contact with the eyeball. In such conditions and when the detachment is an unusually steep one, the perforating coagulation of Šafář seems preferable.

The main objection to Šafář's method is that when I use apparently the same current and the same technic I get a great deal more coagulation in one case than I do in another, possibly owing to peculiarities of the individual tissues, or to certain physical factors which cause variations in electrical action. The goal is to cure the detachment with as little trauma as possible to the delicate structures with which one is dealing.

Transient Fluctuations in the Scotoma of Glaucoma. Dr. John N. Evans.

One hundred and forty-three carefully controlled studies of glaucoma simplex were presented and the statistics set down in tables, and also in the form of a graph which showed the area of the scotoma, the tension and the visual acuity. This permitted direct comparison of the three, and made evident the importance of examining glaucomatous patients always at the same time of day and under standard conditions which can easily be controlled.

DISCUSSION

DR. RALPH I. LLOYD: This paper emphasizes the fact that attention to detail, even in the care of patients with glaucoma, is essentially the price of success.

DR. CONRAD BERENS: I agree with Evans' theory in regard to the cause of transient fluctuations in the size of the scotoma of glaucoma. His simple, inexpensive method of determining the total area of the scotoma is valuable both for scientific and for clinical studies.

If Samojloff's opinion (*Klin. Monatsbl. f. Augenh.* **69:** 59, 1922) is correct, i. e., that in sinus disease the size of the scotomas is not changed by miotics, it is important in differential diagnosis.

Dr. Evans' findings are based on the assumption that variations in the intra-ocular pressure may modify the scotoma. Wegner was unable to produce scotomas in normal eyes or to cause an increase in the scotoma in more than one of five patients with glaucoma simplex by raising the intra-ocular pressure to 50 mm. of mercury. Does Dr. Evans explain the inconsistency of the findings by assuming that the factor, other than the sudden elevation of intra-ocular pressure, is the lowering of tone of the nervous elements by toxins or by the long sustained pressure?

DR. JOHN N. EVANS: I cannot answer the question that Dr. Berens brought up because it would take too long. The work of Wegner and Samojloff was poorly controlled; they did not have enough data to judge whether the changes were due to the experiments or to some of the other factors which I have tried to rule out.

April 16, 1934

The following abstracts and discussion should have been included in the report which was published in the October issue (**12:** 621, 134):

ANISEIKONIA: DIFFERENCES IN SIZE AND SHAPE OF OCULAR IMAGES. DR. JULIUS F. NEUMULLER.

By aniseikonia is meant differences in the size and shape of the ocular images of the two eyes. This condition may be caused by an optical or by an anatomic factor, or by a combination of both. The optical factor consists of the anisometropic properties of the two eyes, and of the shape and power of the ametropic corrective lens. Such unequal optical properties create either over-all, meridional or otherwise unequal optical retinal images. The anatomic factor may arise in an unlike arrangement of the retinal neuro-epithelium or in the components of the visual cortex. An image covering more units in one retina than in the other will be recognized as a larger image.

Aniseikonia producing a functional discrepancy between the two eyes creates grave binocular disturbances. From a foveal discriminatory ability of 10 seconds and the conventionally accepted peripheral dis-

crimination for disparate images, it has been deduced that aniseikonia of less than 0.25 per cent should be measurable. Clinical tests have revealed that in the average patient aniseikonia of this magnitude can be measured.

The correction of aniseikonia is effected by iseikonic lenses. Purely iseikonic lenses lack effective power but have specified magnification. Just as a Galilean telescope consists of plus power objective and minus power ocular, separated so that their respective focal points coincide, in a similar manner the iseikonic lens may be considered as consisting of a planoconvex lens (ocular surface) separated by a layer of glass. The thickness of the lens is arranged so that the respective foci of the surfaces of the two lenses coincide. Iseikonic lenses may be applied in the form of "fitover" lenses in front of the ametropic corrective lenses, or the iseikonic may be combined with the ametropic correction. In a certain percentage of cases the aniseikonic-ametropic corrective lenses assume double lens form consisting of two single lenses cemented together so as to be air-tight at the edges, a form used to gain optical and cosmetic advantages.

Investigations have disclosed that ophthalmic lenses in general distort spatial perception, depending on their base curves, power and location before the eyes. Prisms particularly tend to increase the retinal image when the visual axis approaches the apex of the prism. By proper choice of base curve, thickness and adjustment, these distortions can be eliminated.

Aniseikonia is determined by a specially designed instrument called the ophthalmo-eikonometer. Since the ametropic and the aniseikonic conditions are closely interrelated, it has been found necessary to incorporate a refraction instrument in the ophthalmo-eikonometer. This subjective refraction is performed monocularly under single binocular fixation. The patient perceives binocularly the distance or near target screen and moves a spot of monochromatic red light along a side arm. These spots of light are reflected by means of 45 degree semitransparent mirrors into the eye under test. The sharpest setting of these spots of light denotes in diopters the spatial location of the conjugate retinal point. Thus the ametropic spherical astigmatic correction can be read off, and the location of the conjugate monocular retinal points in space can be equalized in reference to the single binocularly observed distance or near screen. This method of determining the refractive condition discloses for each eye its hyperfocal interval, which is the distance through which perfect vision is maintained without any change of accommodation.

EXAMINATION AND CORRECTION OF ANISEIKONIA. LEO F. MADIGAN.

A conspicuous difference in the size of the retinal images of the two eyes has been recognized in cases of unilateral aphakia and in those of marked anisometropia, but in the past discomfort has not been attributed to smaller differences in the size of the retinal images. There is a group of patients with aniseikonia who complain of discomfort and fatigue after correction has been made for refractive error and muscular imbalance. The discomfort may relate to the eyes in symptoms of

asthenopia, or it may be interpreted in terms of neurologic or gastrointestinal symptoms.

The test for aniseikonia is made by means of a large screen placed 20 feet (6.09 meters) from the instrument for the distance test, and a smaller screen placed at 16 inches (40.6 cm.) for the near test. At the center of the screen there is a black disk which is seen by both eyes. At 4 degrees from the center of the disk in each of the four cardinal directions there is a small hole in the screen. From the rear of the screen light is projected through these holes at such an angle that it is visible to one eye only. The other eye sees the holes as black dots. Thus the patient in looking binocularly at the screen with one eye sees the black disk with four illuminated dots and with the other eye sees the black disk with four black dots. The black disk is fused, but the dots are not. With the same origin, each set of lights and dots should subtend equal angles at the nodal points. By comparing the position of the lights relative to the dots, the relative size of each retinal image is ascertained.

Measurement of the amount of aniseikonic difference is made by use of adjustable iseikonic lenses, and is given in terms of increase or decrease of one image necessary to make it relatively equal to the other image. Change in the size of the image is accomplished by changing the separation of the plus and the minus lens in the iseikonic combination, an adjustment which is read directly from a calibrated drum on the cell holding the lenses.

In connection with the effect of change in the aniseikonic condition with change in the dioptric condition, mention should be made of the change in the size of the image due to the use of power lenses. Every power lens may, potentially, change the size of the retinal image in the eye before which it is placed. In general, a plus lens increases and a minus lens decreases the size of the image. The amount of change in magnification varies with the power, the base curve and the location of the lens used. For instance, a plus 1 diopter sphere will increase the size of the image 2.3 per cent if the lens is flat, and 3.3 per cent if the lens has a base curve of 6 diopters.

Correction of refraction and aniseikonia is accomplished by the use of iseikonic lenses. In some cases a single lens before each eye suffices. In other cases a single lens may be required before one eye and two lenses before the other. The two lenses are cemented together at the edges and are held by the single eyewire of the frame.

It is not to be expected that all patients with aniseikonia require correction, since there seems to be a tolerance for aniseikonia just as there is for ametropia. The average difference in size of retinal images in normal persons free from symptoms was found to be 0.53 per cent. The average difference in size of retinal images in patients with symptoms was 1.53 per cent. Aniseikonic correction has been prescribed for more than 500 patients, in 80 per cent of whom satisfactory results have been obtained following the correction. Relief from symptoms has been obtained following the correction of disparity in size of retinal images as low as 0.75 per cent. Some of the patients have been wearing aniseikonic corrections for four and a half years.

DISCUSSION

DR. ELMER H. CARLETON, Hanover, N. H.: It is a common occurrence to hear patients say that they have been served successively by reputable oculists and yet, to obtain comfort, they have been compelled to restrict their reading. After having spent considerable time in Professor Ames' laboratory at his invitation, and after having seen results in patients who cannot be placed in the neurotic class, I am convinced that there is evidence of value in the method. The work has advanced to such a stage that examination and treatment for aniseikonia should be considered in a considerable percentage of cases of ocular disturbances.

DR. WENDELL L. HUGHES: A number of patients have been examined at New York University during the past few months. Although it is too early to draw definite conclusions, certain observations have been made which confirm the experiences of Professor Ames' staff at Dartmouth College.

1. There is a considerable proportion of the wearers of glasses, about 30 per cent, who have aniseikonia, and the majority of these patients have symptoms therefrom.

2. A method of measuring the relative size of each retinal image has been devised through the leadership of Professor Ames and his staff.

3. Lenses have been devised to equalize the sizes of the retinal images.

4. Iseikonic lenses may be expected to afford relief from symptoms in about 80 per cent of the wearers.

5. The time necessary to complete the examination, from one to four hours, constitutes the chief objection to the method. The cost of the necessary equipment, the time consumed in examination and the cost of the lenses make the procedure somewhat expensive.

PROFESSOR ADELBERT AMES JR., Hanover, N. H.: It is known that the relative size and shape of the retinal image are subject to change, but it is not known what the physiologic functions are which produce these changes. Are they due to alterations in the dioptric images formed at the retina, or are they due to alterations in the distribution of rods and cones, or to both? What is the correlation between aniseikonia and cyclophoria? Is aniseikonia a factor in the etiology of progressive myopia or of migraine?

Evidence is already at hand that aniseikonia may be an important factor in squint. It can be demonstrated that binocular vision is practically impossible with aniseikonia of more than 4 per cent. Measurements in a number of cases of squint have shown an aniseikonic difference of more than 5 per cent in every case. Until cases of squint are found in which there is no aniseikonia, the presumption is that this condition is an important factor in squint.

Even slight amounts of aniseikonia derange the stereoscopic interpretation of the objective field, leading to false estimation of the positions of objects. A difference in the relative sizes of retinal images of 3 per cent will cause a vertical plane viewed at a distance of 20 feet to appear tipped through 70 degrees as interpreted by binocular vision. In the present stereoscopic test for aviators this fact is not taken into consideration, and may explain why some licensed aviators always land with one wing lower than the other.

GERMAN OPHTHALMOLOGICAL SOCIETY

Fiftieth Annual Meeting, Heidelberg, Aug. 6 to 8, 1934

DR. H. ARRUGA, *Chairman*
BARCELONA, SPAIN

TRANSLATION BY PERCY FRIDENBERG, M.D.
NEW YORK

(*Continued from p. 512*)

Scientific Session, August 7

TWO UNUSUAL CONJUNCTIVAL DISORDERS. DR. VOM HOFE, Cologne.

Cystic Degeneration of the Conjunctiva.—A woman, aged 20, presented a multiform exudative erythema of unexplained origin and an acute pseudomembranous conjunctivitis. After the acute condition had healed, there was an extensive cystic degeneration of the conjunctiva, accompanied by peculiar changes involving the finger-nails. The cause of these changes was not determined. They may have been due to disturbances of internal secretion, as the basic metabolism was somewhat increased (demonstrations).

Chronic Pseudomembranous Conjunctivitis.—In this case there was unilateral involvement of the upper tarsal conjunctiva. Bacteriologic and constitutional examinations gave negative results. The membrane had been present for months. The final outcome was organization so that a firm cartilaginous layer remained (demonstrations).

DISCUSSION

DR. LÖHLEIN, Freiburg: The cystic spaces in the first case are, in my opinion, to be attributed to disturbances in the position and arrangement—to displacement if not actual transportation—of superficial epithelial structures. This occurs quite frequently with destructive and degenerative processes of the conjunctiva. In the second case, it seems to me, if I may say so, that one may be dealing with an artefact. I have seen a similar case of extremely severe membranous conjunctivitis in which culture and smears showed only Bacillus xerosis. In order to decide the question which naturally suggested itself at once as to the possible presence of real diphtheria bacilli, the Greifswald bacteriologist, Löffler, was consulted, who, as the discoverer of the diphtheria bacillus, must certainly be considered the highest authority. He could not decide whether true diphtheria bacilli were present or not. Two days later, however, grains of sand were found in the patient's upper culdesac and it was possible to prove self-mutilation as the underlying obscure disease factor.

DR. BARTELS, Dortmund: In all such cases of remarkable or unusual conjunctival disease, particularly in women, one must think of the possibility of simulation or aggravation and of artefacts produced by self-mutilation. This applies especially to the second case. In the first case, there might also be considered a possible proliferation of glands which are scarce in this region in man but very often noted in swine, as described by Strohmeyer.

DR. CLAUSEN, Halle: Vom Hofe's first case seems to me to be one of atypical pemphigus. The bullae may be very tough and persist for many months. In one case of severe pemphigus I observed quite similar lesions of the conjunctivae and, particularly, pathologic changes involving the finger-nails which were strikingly similar to those shown on the photographs presented here.

DR. VOM HOFE, Cologne: The cysts might well be formed by transportation of epithelial cells into the deeper layers. There was not the slightest question here of any artefact, as by self-mutilation. The pathologic lesions of the skin and nails alone speak positively against such a possibility. And it certainly was not a case of pemphigus, which produces bullae that quickly rupture, not cysts that persist for months. Besides, the ruptured cysts leave as residua only little superficial erosions and no cicatrices except, of course, the strands formed by symblepharon. The second case, too, cannot have been caused artificially. Why was the eye always free from irritation? Why were the lesions limited strictly to the conjunctiva of the upper lid? Finally, in such a case one would have noticed occasional changes for the worse. Besides, this patient was kept under observation in the clinic for a long time. I maintain that here, too a sufficient explanation for these two conditions has not been forthcoming.

RÔLE OF THE VORTICOSE VEINS IN THE PRODUCTION OF AN ACUTE ATTACK OF GLAUCOMA. DR. SCHIECK, Wurzburg.

Microscopic sections from an eye that went blind from acute inflammatory glaucoma after the disease had, at first, run the course of glaucoma simplex are shown. Fourteen days after the transition to the inflammatory phase, the eye had to be enucleated, as miotics, an epinephrine substance, and like drugs, and finally an iridectomy had not succeeded in relieving the attack. Histologic examination showed an extraordinary dilatation of the choroidal vessels, especially where they debouched into the vorticose veins, together with a relative narrowing or even obliteration of the lumens of these veins. There was no trace of disease of the walls of the veins, and just as little evidence of mechanical compression through pathologic changes in the wall of the sinus giving exit to the vorticose veins, so as to obstruct the vessels at their origin (Heerfordt). The one and only pathologic factor to be considered was the increased intra-ocular tension acting on the sclera and compressing the vorticose veins in their oblique passage through it, thus causing the serious "hemostatic" disturbances in the choroidal circulation. The anterior regions of the uvea also showed markedly dilated veins. If iridectomy does not succeed, by opening up the filtration angle, in lowering tension to the point of relieving the pressure on the sclera, and allowing the vorticose veins to open again spontaneously, one has the well known clinical picture of malignant glaucoma, and that generally means enucleation.

CAUSES OF DIURNAL VARIATIONS IN INTRA-OCULAR TENSION: A SIMPLE PROCEDURE FOR THEIR DETERMINATION IN GLAUCOMA. DR. POOS, Münster.

Among the factors inducing a nocturnal rise in intra-ocular tension, special attention has been paid to the alteration in the blood volume

(venous congestion in the regions of the head) due to the horizontal position and to the putting of the eyes at rest during sleep (loss of massage action by extra-ocular and palpebral muscles). Another significant factor acting locally is the tonic contraction of the orbicularis muscle, which is kept up steadily with closed lids, and the intensity of which varies as sleep is deep or light and also with the degree of illumination from without. In this way, an intra-orbital increase of pressure is produced which weighs on the venous exit channels of the globe. In a large series of eyes, some normal as to intra-ocular tension and others with primary or secondary glaucoma or with uveitis showing a tendency toward increased tension, these factors were studied in forenoon office consultations by means of the lid-closure or sleep test. For this purpose the patients, after having been out of bed for several hours and having just entered the office, were made to lie down and rest with their eyes closed for from thirty to forty-five minutes. Normal eyes reacted to this physiologic provocative test with hardly any increase of tension, and some, indeed, with a drop in tension (this in patients who were unable to keep their eyes quietly closed without moving the globe and/or repeatedly winking the lids). Of 31 eyes with primary glaucoma, 70 per cent reacted with an increase of tension of as much as 15 mm. of mercury. In an attack of glaucoma (status glaucomatosus) the forenoon sleep test was always strongly positive, and the maximum tension noted at the early morning measurement was reached within forty minutes. Of 10 patients with secondary glaucoma, 6 responded with increased tension, up to 22 mm. of mercury; 2 showed unchanged tension, and 2 a drop. Of 25 eyes with uveitis and increased tension, almost all reacted positively. In one case tension rose from 29 to 57 mm. of mercury. In the ordinary routine it is not feasible to plot a curve of the diurnal variation in most cases, and there has been no general acceptance of the more complicated physiologic provocative tests, such as venous stasis of the cephalic circulation, drinking, caffeine, paracentesis of the anterior chamber, the gravitation test and so on. The physiologic provocative test by simulated sleep (lid-closure test) up to about one hour has its failures, like any other test, but makes up for this by the advantage of being exceedingly simple for both the surgeon and the patient. As this test allows one in all positive cases to draw quite sufficient conclusions as to the nocturnal increase of tension to be expected in spite of pilocarpine and physostigmine, this simple method offers, especially to the practitioner, a means of greatly extending the diagnostic possibilities in glaucoma and, above all, in preglaucomatous states.

A New Operation for Glaucoma. Dr. Sondermann, Berlin.

It seems justifiable to propose a new operative procedure for the reduction of increased intra-ocular tension without having been able, so far, to test it out sufficiently in practice. The procedure is based on the theory that the primary cause of increased tension in simple glaucoma is generally to be sought for pathologically in an embarrassment of the venous off-flow from the eye through the venae vorticosae in direct consequence of pressure from a sclerosed sclera. Its aim is to loosen up the sclera and to facilitate the venous off-flow from the uveal tract by the

formation of new veins between this tract and the conjunctiva. After radial incision of the bulbar conjunctiva a spatula-like knife is inserted in the sclera very obliquely about 3 mm. from the limbus and is carried forward and laterally to the suprachoroidal space, forming a canal from 2 to 2.5 mm. wide, into which a narrow strip of conjunctival tissue is introduced. In severe cases, this procedure is repeated at several points.

DISCUSSION OF PAPERS READ BY DR. SCHIECK, DR. POOS AND DR. SONDERMANN

DR. VOM HOFE, Cologne: The effect of sleep and that of lid closure are surely not identical. Under Löhlein in Jena a large number of night measurements were made at regular intervals in cases of glaucoma. Most of the increased tension was noted in the early morning hours. Undoubtedly many factors are associated in bringing this and similar reactions about. For one thing, the vegetative functions, e. g., respiration and circulation, run a different course during sleep and the waking state. At any rate, the provocative test offered by Poos is a valuable addition to procedures for the diagnosis of glaucoma.

DR. LINDNER, Vienna: In reference to the article by Poos, Dallmann, in his day, reported on similar tests but attributed the rise in tension to the absence during sleep or when the lids are kept closed of eye motions which act in the way of massage to lower tension. It should be a simple matter to determine whether orbicularis pressure has the effect postulated by Poos, which I think unlikely. Alcohol akinesia lasting for several days would be sufficient to settle this point. In reference to Schieck's article, even under normal conditions, there is a certain degree of strangulation of the vorticose veins at their exit from the sclera, producing a physiologic stasis. Furthermore, it has been an accepted theory for a long time that the vortex veins which run obliquely through the sclera are hindered in their function by an increase in tension, and that this is accompanied or followed by dilatation of the anterior ciliary veins as well. Hence, I believe that the pictures presented by Schieck could be found just as well in cases of chronic glaucoma, and that they are, accordingly, by no means characteristic of, or pathognomonic for, acute inflammatory glaucoma. In regard to the management of hopeless states of acute glaucoma, I wish to remind the members of my communication made to this society a year ago, on operative fistulization in the posterior portion of the globe. This procedure generally gives a deep anterior chamber and a reduction of intra-ocular tension.

DR. BARTELS, Dortmund: Schieck has considered only the efferent blood vessels. The afferent arteries, too, show most remarkable pathologic changes. The anterior arteries present a marked narrowing of the bore, while the posterior arteries lose their muscularis in going through the sclera. As a matter of fact, one never has an entirely "fresh" case of glaucoma, and in every instance it behooves one, in interpreting the data, to ask oneself what is primary, i. e., causal, in glaucoma, and what is secondary or resultant from glaucoma. At present it does not seem possible to discriminate clearly. One may also find endothelial proliferations in the vorticose veins, a condition which I described at the time.

DR. JESS, Giessen: Ten years ago a practitioner-oculist had the bright idea of curing retinal detachment by transplanting strips of conjunctiva into the interior of the eye by way of a scleral incision. I had occasion to examine a patient a long time after this operation had been performed and had ended in failure. A dense fibrous cicatrization had taken place without a trace of vascularization. In addition, a complicated cataract had developed.

DR. COMBERG, Rostock: Does not the mydriasis which naturally accompanies these tests constitute a complicating and possibly misleading factor in view of the findings demonstrated by Seidel?

DR. SERR, Heidelberg: I wish to stress the remarks of the previous speaker in regard to Poos' tests. In sleep there is constantly a miosis, whereas in Poos' test of resting with the eyes closed there is as regularly a mydriasis. This fact is of something more than theoretical significance. The effect of the miosis of sleep on the variations in tension of suitable glaucomatous eyes with reaction to light and shade was demonstrated by me before this society a number of years ago. Hence I find that essential and indispensable control tests were lacking in the tests described by Poos.

DR. JUNG, Cologne: Jess has mentioned a practical oculist who suggested a procedure for the treatment of detachment of the retina much like that presented to this society by Sondermann. But long before, a person who shall be nameless had advised inclusion of conjunctiva under the sclera as an operation for glaucoma, and had tried to get some cheap publicity for himself in the daily press. I was able to spike his guns by an article in an influential political journal carrying the challenge that if he really had an infallible cure for glaucoma, he would have laid his contribution on the altar of science for a final judgment and made a communication to our society on the matter.

DR. LÖHLEIN, Freiburg: I was able to operate in 2 cases of painful absolute glaucoma according to the original suggestion of Sondermann. Unfortunately I was able to follow up these patients for only from one to two months. At that time there was, in one case, freedom from pain together with regulated tension, and there was evident neoformation of blood vessels in the trephined and cauterized area. At Greifswald, before the World War, I encountered an infection after one of my early Elliott trephine operations, and this induced me to transfer the site of trephining to the posterior section of the sclera, which is not in danger of infection. I did this in a number of cases but only succeeded in getting a transitory drop in intra-ocular tension, although I took the trouble in one case to line the trephine opening with a free conjunctival transplant in order to get permanent epithelization of the canal. Possibly one might do justice both to Schieck's findings and Sondermann's theory by shaving away some lamellae and thus thinning down the outer wall of the sclera in the immediate neighborhood of the exit of one or more vorticose veins.

DR. WESSELY, Munich: The rôle of the vortex veins in acute glaucoma has had attention for a long time, and just as in the case of the narrowing of the filtration angle when that was the *punctum saliens* in the study of the causation of increased intra-ocular tension, the main difficulty has been to answer the question as to what is cause and what

effect. To what degree is the compression of the vortex veins in their course through the thickness of the sclera due to increased intra-ocular tension, and, *pro tanto,* secondary? How far, on the other hand, is this process itself the cause of a rise in tension which may, under certain conditions, be uncontrollable? This dovetailing of cause and effect might be particularly difficult to unravel (*sit venia verbo*) the moment one has to deal with a case of so-called malignant glaucoma, in which the momentary overfilling of the uvea with blood immediately following the operative procedure adds another complicating factor to be considered in the interpretation. It will be most interesting to hear, when Schieck closes the discussion, more in detail and more explicitly, what conclusions the case so carefully observed and so completely presented allows one, in his opinion, to draw in the interpretation of this particular aspect of the question.

DR. VAN DER HOEVE, Leyden: Schieck's interesting remarks show that congestion in the vorticose veins is of great significance in the development of malignant glaucoma, and that in such cases the tapping (*Absaugung*) of the vitreous might be effective. It may be well to remember the old theory of Weber as to the pathologic mechanism of malignant glaucoma. According to this hypothesis, the lens is crowded forward, engages itself in the scleral groove and is held fast there, so that filtration in the angle of the anterior chamber is permanently arrested. In cases of acute glaucoma in which blindness persists in spite of an iridectomy, or in which one wishes to preserve the already blind eye in spite of its loss of function, van der Hoeve advises an Elliot trephine operation followed immediately by a posterior sclerotomy in which a small amount of vitreous is allowed to drain off. A blunt spatula is then introduced through the trephine opening, and the lens is pushed backward. In this way both etiologic factors are canceled.

DR. SCHIECK, Wurzburg: Both anterior and posterior ciliary arteries in my case were dilated and congested. Eyes which have become blind, especially in malignant glaucoma, cannot give one any clue to the mode of the increase in intra-ocular tension even as a primary factor.

DR. POOS, Münster: As to Vom Hofe's objection, the results of my experiments did, as a matter of fact, appear to contradict Thiel's observation of a two-phase nocturnal rise in intra-ocular tension. But, as already noted in the paper, many patients with glaucoma show after only from forty to forty-five minutes of the lid-closure test an increase in intra-ocular tension corresponding to the acme of the early morning measurement. This is explained by the fact that the tension of the orbicularis muscle which accompanies the lid closure of awake patients in the morning office tests in bright illumination is more active and more effectual than it is when patients are really asleep at night in the dark. It must also be considered that general blood pressure sinks at night during sleep, and that in awake patients in the daytime blood pressure is higher when they are reclining than when they are sitting up, and higher when they are sitting up than when they are standing. So that in the series of "sleep tests" instituted by me the sleep factors, accordingly, were intensified in their action. In answer to Lindner's observations I should like to point out that, previously, in my paper, I mentioned Sallmann in connection with those authors who assume that keeping the

eyes at rest is the cause of the nocturnal increase in tension. I, too, am of the opinion that putting the eyes in a position of complete and lasting rest is, as mentioned in my paper, one of the sleep factors in the causation of increased intra-ocular tension. As to objections raised by Comberg and by Serr, respectively, in regard to the pupil as a sleep factor, this does not enter into the problem in nocturnal rise of tension since there is continued miosis during sleep. But in my investigations with the lid-closure test, as well, the question of width of pupil does not play a rôle, at least certainly not in the great majority of cases. In a few exceptional cases in which the anterior chamber is very shallow this factor may have some significance if the pupil dilates. Changes in the width of the pupil certainly did not play any etiologic rôle in the numerous eyes I studied which presented uveitis with plus tension and which were kept continuously under the influence of scopolamine, nor in glaucomatous eyes, which were kept under pilocarpine physostigmine and gave a positive reaction.

DR. SONDERMANN, Berlin: In answer to Jess, I wish to state that in the case which I cited there was normal intra-ocular tension or even, possibly, slight hypotony. In such a case one can hardly expect new venous vessels to form in the eye, as there is no abnormal stasis in the vorticose veins and the latter, moreover, function normally. Neoformation of vessels is only to be expected when the vorticose veins have been subjected for some time to abnormal pressure and no longer function normally, as in cases of simple glaucoma. In Löhlein's experimental studies and those of the colleague mentioned by Jung, there was an endeavor to drain fluid from the interior of the eye as in the fistula-forming operations involving the anterior chamber, but there was no formation of new vessels. This conception could have been formed only after I had proposed my theory. I am also skeptical as to whether the thinning of the sclera over the vorticose veins, a feature mentioned by Löhlein, is of any practical causal significance. This factor is purely hypothetical for the one reason alone that it is difficult to determine the exact course of these veins. The stasis in the uveal vessels found by Schieck in acute glaucoma I take to be a secondary manifestation in the nature of a complication or a sequela. I am of the opinion that, in an eye predisposed to acute glaucoma, vascular disease involving the arteries as well as the veins is in the forefront of the process. As a result the circulation of the blood, as well as that of the aqueous, is reduced in volume, but also, and for the same reason, the function of the efferent channels in maintaining a condition of equilibrium of fluid exchange in the eye is lowered. If under such conditions the blood stream rises under the stimulus of excitement or the influence of alcohol and the secretion of aqueous is also increased thereby, the regulating mechanism cannot meet the intensified demand. This may be due either to the fact that the iris, which in these cases is generally more rigid than usual, does not allow a sufficiently rapid off-flow of fluid into the anterior chamber or to the fact that the exit of fluid is so slowed at the filtration angle that the root of the iris bulges forward and obstructs the filtration angle, a feature on which Elschnig, particularly, has laid stress. In this way a vicious circle is started for a sudden rise in intra-ocular tension. The process of the increase in tension in malignant glaucoma after the anterior chamber has been opened is different at least to this extent that, in my

opinion, one is dealing with an eye in which, besides the blood perfusion of the eye, the secretion and filtration of aqueous are so reduced that, after the anterior chamber has been abolished, there is no reproduction of aqueous and, accordingly, all adjustment of the relations between blood inflow and blood outflow must cease.

STRUCTURE OF THE VITREOUS. DR. LINDNER, Vienna. .

The studies of Baurmann and Thiessen demonstrated that the vitreous has a fibrillary ultramicroscopic structure, but did not settle the question as to whether there was any coarser structure. Baurmann contends that there is not, while Lindner holds to a lamellar structure of the vitreous cortex. To begin with, he shows that there is no basis for the widely accepted view or rather assumption that the vitreous structure seen in histologic sections is a result of coagulation-precipitation pure and simple. In the first place, the fibrils of the vitreous are disposed in such a way in the histologic section that they cannot be explained as due to the aforementioned physiochemical process. This statement applies particularly to two places: the base of the vitreous and the region immediately behind the lens. In eyes specially prepared for the purpose it is possible to produce *Liesegang* circles (*Ringlinien*) which show clearly that the diffusion lines in these two regions have a course quite different from that one must anticipate if one accepts the coagulation-precipitation theory as an explanation of the appearance of vitreous fibrils. Accordingly one must conclude that the structure of the vitreous as seen in the microscopic specimen is conditioned predominantly by the histologic structure of the vitreous which underlies it. Furthermore, that one may infer a lamellar structure in the vitreous follows from the observation of deposits of such elements as red blood cells or pus corpuscles in the cortex of this body. I may remind you of Fuchs' work on hemorrhages into the vitreous, and I shall show photomicrographs of sections with infiltration of the vitreous back of the lens which can be explained only on the basis of a lamellar structure of the vitreous cortex, especially since Comberg with the slit lamp demonstrated in the living eye a lamellar arrangement of hemorrhages in hernia of the vitreous. Duke-Elder and Heesch were the first to attempt to explain the discrepancy between the picture of microscopic sections and that of ultramicroscopy. According to these authors, the fixing fluids do not destroy the elementary fibrils of the vitreous but merely cause them to form into fascicles, so that the histologic picture is easily explained.

LOCALIZATION OF INTRA-OCULAR FOREIGN BODIES BY GRUDZINSKI'S METHOD. DR. DZIEGIELEWSKI, Posen.

The method described is a "physiologic" one based on the change of position of the shadow cast by the foreign body on the x-ray plate with, generally, four different directions of gaze of the eye under examination. The patient lies with the side corresponding to the affected eye downward and close to the film-holder, which is 5 cm. from the eye. In front of the eye at a distance of 96 cm. an axis cross is placed with the fixation objects, which are 1 meter apart. The focus of the x-ray tube is 60 cm. away from the plate. The patient's head is supported by a

wooden head-rest allowing the film-holder to be moved easily with each change in the four different directions of gaze. Four exposures are then made with the head in one and the same position, the gaze being directed up, down, left and right. The essential part of this method depends on the change of gaze direction, being exactly 27.5 degrees in each new fixation. It is only when this condition is fulfilled that the change of position of the foreign body's shadow on the x-ray plate corresponds exactly to that of the actual foreign body in the eye. The shadow of the "downward" plate is transferred to the "upward" plate; the shadows of the "left" plate to the "right" plate. The film is then cut up so that on one half of it there remain the "up-right" shadows together with the transferred "down-left" shadows. With a transparent celluloid diagram of the eye, this half of the film is then used for localization. The celluloid plate shows three cross-sections of the globe: (1) sagittal. (2) equatorial and (3) through the meridian. In order to determine the position of the foreign body in profile, the profile picture is placed perpendicular to the line of sight on the x-ray picture of the "up-down" shadow pair in such a way that the "up-down" shadow falls exactly in the middle of the profile picture. The shadow with gaze directed upward will then show the actual position of the foreign body in the eye in profile. The second shadow pair, "right-left," allows one to construct the degree of longitude expressed in hours of the dial. Graphic representation shows the foreign body in the eye projected from in front on the equatorial axis. The degree of latitude in which the foreign body lies is then determined. Again the position of the foreign body from the profile on the anteroposterior axis is transferred to the plane of the hour-circle, and the projection of the foreign body from the middle point of the anterior picture is transferred, likewise, to the axis of the equator. The point of intersection of the perpendiculars together with the middle point gives the degree of latitude of the foreign body. The localization does not take more than two minutes. This method has been in use in the University Eye Clinic of Posen for the last four years and has invariably been found reliable and accurate. It has been found especially serviceable in localizing nonmagnetic bodies, and in every instance the foreign body which had been previously localized was successfully removed with grasping forceps which had been introduced "blindly." This method is recommended because it is simple in operation, calls for only simple appliances which any one can make for himself and gives at the same time exceedingly accurate and reliable results.

LOCALIZATION OF RETINAL POINTS BY OPHTHALMOSCOPIC TRANSILLUMINATION, WITH A NOTE ON LOCALIZATION OF INTRA-OCULAR FOREIGN BODIES. DR. LINDNER, Vienna.

I wish to call attention to the fact that a method of localization which I was the first to work out has been credited to Guist in two foreign journals. I should like to straighten this matter out for the record. Today it almost seems as if one does not require an exact localization. That is not the case. In operations aiming at a walling off (*Abriegelung*) of the tear and/or the detached area, the extent and depth of this walling off must be correctly adjusted. But in Weve's operation an absolutely exact localization is essential. I shall mention briefly the

difficulties encountered in localizing, under ophthalmoscopic control, electrocoagulation points. . . . With this introduction I shall now describe an accurate and simple procedure for localization which requires no special localizing instrument other than the May or Simon ophthalmoscope and which is carried out during the operation. The procedure depends on producing, by means of a diaphragm, a small, sharply limited area of ophthalmoscopic illumination which glows through the wall of the globe. While the operator observes ophthalmoscopically the defect in the upright image, his assistant marks, at the same time, on the outer surface of the sclera the luminous spot which shines through. In cases in which no previous operation has been done one can get as far back as 20 mm. from the limbus. A similar procedure was also used by Weve, but only incidentally as a sort of control in connection with his own method of localization. Furthermore, the operator can check up on the accuracy of his assistant's marking in flat detachment by having him grasp the sclera at the luminous point with a pair of fine forceps and make slight pressure. The operator will then see the indentation in the fundus with the ophthalmoscope. Whether this method based on ophthalmoscopically visible indentation is practical in the case of fundus points situated far back is a question which I cannot answer as yet, since my experience as to this detail has, so far, not been sufficient. At all events, the procedure which I have presented is the best and most accurate method of localization so far offered. I shall now report 2 cases of suspected intra-ocular neoplasm in which I was able, after this schematic localization, to remove part of the suspicious growth for microscopic examination. . . . I shall now describe a new method for the localization of nonmagnetic foreign bodies. It consists in exposing the choroid over a large area and then placing a Lange lamp in contact with the opposite pole of the globe. A dense foreign body will then cast a shadow on the exposed choroid.

MEASUREMENTS OF THE FUNDUS OCULI. DR. LOBECK, Jena.

Accurate quantitative determinations are of practical importance in determining the caliber of retinal arteries if one is to draw any conclusions as to the patient's general condition. It is essential to know the proportion between the calibers of arteries and those of veins and between the calibers of vessels and the papillary diameter, the latter of which has, quite independent of refraction (? P. H. F.), a constant value of 1.5 mm. In order to base one's interpretation on figures one must have a method which allows one to measure accurately the calibers of the vessels and the diameter of the disk at the same time. I shall briefly report on previous experiments by various authors. . . . I shall now decribe an apparatus which meets all the demands. The ocular of a simplified Gullstrand ophthalmoscope is divided by a meridional section into two halves which are adjustable in relation to each other by a micrometer screw. Besides this, the ocular as a whole can be rotated around its optical axis. The measurement is carried out as with a heliometer, the operator approximating the partial pictures of the fundus corresponding to the two halves of the lens by turning the adjusting screw until the contours of the object to be measured touch at only a single point. It thus becomes possible with the utmost ease and

in the shortest possible time, even with an undilated pupil, to measure the calibers of the retinal vessels and the diameter of the disk in reciprocal terms of relations under simultaneous and uniform optical conditions. This arrangement has certain advantages over Henrikson's procedure, which consisted in dividing the ophthalmoscope lens—not the ocular—into two halves and then measuring the thickness of the retinal vessels. For one thing, my method allows the measurements to be carried out with an undilated pupil, and allows the papillary diameter to be measured at the same time. This is not feasible with Henrikson's apparatus. It goes without saying that the bisected ocular lens can be used to measure any other objects or distances in the fundus as, for example, retinal tears, in terms of the papillary diameter.

MY EXPERIENCES WITH ELECTRO-ENDOTHERMY IN RETINAL DETACHMENT. DR. LARSSON, Stockholm.

My data go back to 1923 when I began to study the action of electro-endothermy experimentally in rabbits. The aim was to produce an adhesive choroiditis which might presumably have a favorable influence on the healing of a retinal detachment. The first operation in the clinic was not performed until January 1930, and the first report and description of this operative procedure with a detailed complete report of 7 cases in which the operation was performed was published in June 1930 (Larsson, S.: Operative Behandlung von Netzhautabhebung mit Elektroendothermie and Trepanation; vorläufige Mitteilung, *Acta Ophth.* **8:** 172, 1930). At that time there was no way of my knowing that Weve of Utrecht had, about a month previously, made a short communication in Dutch (Weve, H. J. M.: Retinal Detachment: Treatment by Gonin's Method, *Nederl Tijdschr. v. geneesk.* **74:** 2354, 1930) intimating briefly that he was working in the same direction. I am at a loss to understand why my work in this field is somewhat misunderstood just in Switzerland and Holland and in Germany, too. An attempt has been made to show that in the treatment of detachment I do not sufficiently consider the significance of tear. This is not the case. On the contrary, the importance of the solution of continuity of the retina is stressed in all of my works expressly. I am of the opinion that the favorable action of endothermy is due to its causing an adhesive choroiditis which glues or solders the retina to the choroid, and to the closure of any tear present. Extensive endothermy including the region of the tear and touching also the surrounding portions of the choroid has as its object the soldering down not only of the detached portion of the retina but of that portion of the whole retina which might later on become a locus minoris resistentiae on account, possibly, of degenerative processes which had already started. Such a procedure walls off the region of the tear by adhesions even if the tear itself has not been directly touched by the operative procedure. Large portions of the peripheral retina can, however, be brought back to adherence without any danger by extensive endothermic treatment. This has the advantage that one gets around the necessity for accurate localization of the tear, which is always a difficult matter. This procedure is, by and large, the same as that described in June 1930. I shall now give data on 98 cases in which the operation was performed with complete success in 52.

Scientific Session, August 7

DR. LINDNER, VIENNA, *Presiding*

TECHNIC OF DIATHERMOCOAGULATION AND USE OF PYROMETRIC ELECTRODES IN DETACHMENT. DR. MEESMANN, Berlin.

Since 1932 I have used this method in over 150 cases. In the first 50 cases a perfect anatomic and functional result was obtained in 52 per cent. Later there was constant striving for further improvements and particularly to avoid postoperative vomiting and failure due either to copious intra-ocular hemorrhage or to insufficient dosage of diathermy. Postoperative vomiting with nausea lasting from one to seventy-two hours occurred in almost 30 per cent of the patients with retrobulbar anesthesia. On that account, intravenous injection of n-methyl-cyclo-hexanyl-methyl barbituric acid to the point of complete narcosis was substituted. The use of this drug requires special experience. The art of correct dosage is of prime importance. In order to get results with as small a dose as possible it is necessary to give about 0.5 to 0.3 ampule of scopolamine di-hydro-morphinone hydrochloride one-half hour before operation. In the first minute 1 cc. or at most 1.5 cc. of n-methyl-cyclo-hexanyl-methyl barbituric acid is injected. Even in this short time the degree of tolerance for the drug is to be estimated. In the second and third minute a second injection of the same amount is made. The degree of tolerance is extremely variable. Generally, 4 cc. is sufficient to cause deep sleep, but frequently much smaller doses are enough. The narcosis is to be controlled in such a way that the patient is kept just asleep. The total amount of narcotic necessary ranges from 4 to 12 cc. with an operation lasting up to thirty minutes. In only 1 case did motor restlessness come on several hours after operation, probably as a result of a rather generous dose, namely, 13.5 cc. of the narcotic. N-methyl-cyclo-hexanyl-methyl barbituric acid reduced the postoperative vomiting to a minimum. As a rule the patient stands the narcosis well and suffers no unpleasant after-effects. Difficulties are met with at times in children, and then ether narcosis may be required. Disease of the liver is a contraindication to the administration of n-methyl-cyclo-hexanyl-methyl barbituric acid, as this drug is toxicologically neutralized in the liver. For more accurate regulation of dosage with relation to the degree of coagulating effect, pyrometric electrodes were worked out in cooperation with the Sanitas Company, of Berlin. Blunt and needle-pointed electrodes of various thickness and of platinum-iridium and constatan are available. The electrodes are carried into a quartz tube which is bent at an angle in front and is fastened at various lengths in the electrode-carrier, which is made of compressible material (*Press-stoff*). In this way it is possible to regulate exactly the depth of the puncture and the size of the perforation. Opposite the active electrode a similarly constructed thermo-element is introduced into the circuit and submerged in ice-water. This raises the difference in potential to the highest point; after heating, for example, to 80 degrees, it amounts to 2 millivolts. When in use, an electrode of this sort is warmed up by the heat of the surrounding tissues so that the coagulation temperature can be read off from the indicator on the milliampere meter, which is calibrated to hundredths of a degree centigrade. The most suitable temperature lies

rather exactly at 75 C. It was soon ascertained that these measurements of temperature gave a decidedly more accurate criterion of the degree of coagulative effect than any measurement of the intensity of the current itself. Accordingly a more minutely regulated dosage is possible, which allows one, under control of the ophthalmoscope, to attain the desired result with the greatest accuracy. The draining off of subretinal fluid, too, can be attained, almost without exception, by perforation, at least with the thicker needles, so that one can and should avoid trephining with its disadvantageous sequelae such as, particularly, the folding of retinal tissue from being incarcerated in the trephine opening.

DISCUSSION OF PAPERS BY DR. LINDNER, DR. DZIEGIELEWSKI, DR. LOBECK, DR. LARSSON AND DR. MEESMANN

DR. JESS, Giessen: In regard to Larsson's claim to priority in the treatment of retinal detachment with high frequency current, I wish to call attention to the fact that here, as in so many other instances, several authors have independently tried out the same procedure. Without knowing about Larsson's and Weve's experiments, we in Giessen also tried to reattach the retina in animals by means of high frequency currents. A Rumanian author, too, wrote me that he had come to the same method quite independently. In answer to Meesmann, vomiting never took place following our diathermic coagulations although the local anesthesia used was exactly the same as that mentioned by him. We waive complete narcosis in order to have the patient able to move his eye at will and so make the retinal region which is under operation by the electric current accessible to ophthalmoscopic examination and control. If an attempt is made to get the same result by passive movement of the eyeball under the pull of suture threads passed through the external ocular muscles, the pull on the softened globe invariably results in distortion of the cornea, and this interferes with a good ophthalmoscopic view of the fundus.

DR. GILBERT, Hamburg: In connection with the communications of Larsson and Meesmann on electrodiathermy and electrocoagulation I desire to call attention to the results attained with a form of operative treatment which is apparently the direct opposite of the aforementioned thermic procedures, namely, the application of carbon dioxide snow after a preparatory trephining or a superficial lattice-form sclerotomy. In Deutschmann's eye clinic this procedure was used occasionally in earlier days, as was cauterization with tincture of iodine, also after preparatory trephining. Both procedures, but particularly that with application of carbon-dioxide snow, were developed systematically only after the numerous therapeutic surgical innovations inspired largely by Gonin's original operation had given ophthalmologists the incentive to improve and perhaps to develop systematically procedures which they had used long before. I have treated my own patients with detachment by this method, on which Deutschmann had reported (*Arch. f. Ophth. Gesellsch.* [sic]), and with such excellent results that I recommend it warmly and confidently, the more so in that the technic is simple, the procedure does not cause marked reaction, and the apparatus is inexpensive. As to the successful results of operative treatment, the Deutschmann clinic has from the beginning taken a rather

skeptical view of the optimistic figures of the first years during which Gonin's apparatus was used, for percentages of 70, 80 and even higher are not to be counted on in detachment, at least, certainly not for any extended period. One gets a different picture from the statistics which various operators have published more recently. Lindner's clinic, for example, reports permanent reattachment with the Gonin and Guist procedures in from 33 to 44 per cent of the cases. In the 19 cases in which Deutschmann and I have operated in the last year or two permanency of the cure is still an academic question, as only 4 cases have been kept under observation or followed up for two years. Even this period, according to our definite and well based experience, is entirely inadequate to establish definitely the permanence of reattachment and cure. Excluding 2 cases which were considered to be hopeless from the start, there have been 9 provisional cures so far in 17 cases, i. e., a little over 52 per cent, or exactly the percentage reported by Drs. Larsson and Meesmann. Among the 8 cases in which the condition is not yet cured, 5 detachments are still under treatment, and 3 have already shown such marked improvement that they will in all probability go on to complete reattachment without further operative intervention. As to the healing process in the treatment with carbon dioxide snow, stress is laid on the particularly copious flooding of the treated regions of the fundus with pigment, the slight external reaction and the restoration of good function as to vision and visual fields. These good results are corroborated by Bietti's report from the Naples clinic.

DR. ARRUGA, Barcelona, Spain: A word or two as to the prognosis. Up to the present, cases which were old or in which several tears showed were considered particularly difficult of operation and unpromising as to results. In my opinion the most important point is that after the operation the retina should be in apposition with the choroid. The success of the operation for detachment depends on accurate localization and on ophthalmoscopic control during the operation; in other words, on great patience in the dark room and even greater patience at the operating table. As to improvement in the mechanical details of localization and in operative method through the use of complicated apparatus and technic, there is this to be said: The main point is to simplify matters, for it is better even from the humane standpoint to have in place of 50 eye surgeons with an average of 50 per cent successes 500 with a mean of 25 per cent, since it is to be anticipated that later on they, too, will have good results in 50 per cent.

DR. BARTELS, Dortmund: In reference to Lindner's paper on the structure of the vitreous, one should not forget the observation of the late Professor Stilling of Strasbourg: If, after sectioning the eyeball frontally into two equal halves, methylthionine chloride is poured onto the exposed surface, one notes about midway between the middle of the vitreous body and the sclera a faint ring colored blue, in other words, a cleft. This observation seems to have been generally overlooked. In reference to the paper read by Meesmann, my experience proves that vomiting at or after operation can be avoided simply by previously inserting a vasenol suppository. In comment on Larsson's paper I wish to lay stress on the importance of Arruga's remarks, with which I fully agree. The most difficult detachments to handle are those with a large bleb. Is Larsson able to cause these to reattach by means of

simple surface coagulation? In my opinion the best method for treating detachments of this type is still the trephining procedure and cauterization. It certainly has given me the best results.

DR. ENGELKING, Cologne: Formerly I employed surface coagulation, pure and simple, with trephining, as did Larsson, but later on I changed from the latter procedure to multiple needle-pricking (*Stichelungen*), as I was afraid of the results of a sudden escape of retinal fluid through the trephine opening; a consideration which also determined me to give up the Gonin procedure. Still, I think that even today one has better prospects of a definitive reattachment in certain cases with the application of pure surface coagulation, even without any subsequent perforation. Such cases are those of disinsertion at the ora serrata or, on the other hand, of "gigantic" tear formations like the one I demonstrate here in which the tear extended over nearly half of the entire retina. In this case I used simple surface coagulation which, however, completely walled off the sound region from that of the affected tissues. While I do not claim that this detachment is permanently cured—it has been under observation only eight weeks so far—it is at least self-evident that it could not have been tackled at all with Gonin's method. On the other hand, a subsequent trephining or multiple microperforations would, in my opinion, have been dangerous. These considerations lead me, too, to consider Gonin's objection (Gonin, J.: Quelques remarques sur le traitement du décollement rétinien, *Schweiz. med. Wchnschr.* **63:** 1287, 1933) in which he refuses to recognize the coagulation method as one different from his own. But there is no question that here one is dealing with two entirely different procedures, with entirely different prospects of definite healing and different indications for application, even if they happen to coincide in one point, viz., the advisability, according to Gonin, of making the tear harmless. As to Meesmann's observations, my associates and I are rarely disturbed by the patient's vomiting during operation or, in fact, later. So we never found it necessary to have recourse to general narcosis. It is true that we limit the injections of procaine hydrochloride to the region of the orbit in which we are obliged to operate and do not anesthetize the ganglion. Perhaps the vomiting in Meesmann's cases may be due in part to the operative detachment of extra-ocular muscles. I know that this complication is rather common subsequent to operations for squint. In our experience, which in this respect coincides fully with that of Bartels, the cases in which it is most difficult to obtain healing are those in which there is a very deep bleb or pouch, for then we often fail to carry the coagulation at the time of operation up and into the region of the margins of the tear. In such cases I have helped myself out by slightly shifting the surface coagulation or, at least, the microperforations and by extending this procedure so far toward the center of the fundus that, during the operation, the effects of coagulation could be seen with the ophthalmoscope. It is true that this puts a somewhat larger part of the retina more or less "out of commission" for the future, but the prospect of an actual and lasting subsequent reattachment is in my opinion definitely increased by it.

DR. VON HIPPEL, Göttingen: Perhaps I am expected to answer Lindner's criticism of Baurmann in regard to possible discrepancies between the ultramicroscopic and the usual histologic or slit-lamp pic-

ture, respectively, of the vitreous. I have many pictures like those shown by Lindner and also made a large number of them known to Baurmann. He is, accordingly, *au fait* with this aspect of the controversy, too, and will undoubtedly answer Lindner himself when this report appears, finally, in print.

Dr. Clausen, Halle: I have carried out retrobulbar injections in 300 operative cases of detachment without ever noting serious postoperative vomiting. In spite of the very favorable experience with the use of n-methyl-cyclo-hexanyl-methyl barbituric acid in the clinic with which I am associated (cf. Klitzsch: *Klin Monatsbl. f. Augenh.*) I do not recommend it in connection with operations for detachment. During and after such operations it is of prime importance to be able to have the spontaneous cooperation of the patient in directing his gaze in this or that desired direction for the purpose of ophthalmoscopic control. The clouding of the cornea can be quickly eliminated by irrigating with saline solution. My latest procedure consists in most exact and careful localization followed by a preliminary coagulation with the finest needle-pointed electrodes. The area of coagulation is then inspected with the ophthalmoscope and checked up, after which it is feasible to place additional coagulation points quite accurately in the tear and its surroundings. Besides the large bullous detachments, those with marked fold formation give the least favorable results, as here one has very often to deal with adhesions between the folds which are not broken up and cannot be broken up by coagulation. If the retina appears to lie smoothly in contact with the choroid at all points after operation, one may generally count on a good result. At times one can get excellent results with the actual cautery, closing very large tears which would present many difficulties to closure by electrocoagulation. Not infrequently, however, the unpleasant traction folds which are a common result of the escharotic effect of the Paquelin cautery again jeopardize completely the originally promising outlook for a permanent reattachment. I now use electrocoagulation exclusively. A final decision on the merits of this procedure and its possible modifications cannot be rendered at present.

Dr. Mylius, Hamburg: In the operative treatment of detachment one is often obliged, as a consequence either of relapse or of insufficient effect of the first operation, to repeat the procedure in the same area as before. In this one is greatly hampered by the scar tissue resulting from the first surgical intervention. Repeated coagulation, too, is extremely difficult in this area, and much higher intensities of current are requisite. The most unfavorable cases are those in which the margins of the tear are detached and protruding.

Dr. Schnyder, Solothurn: Professor Vogt reported (*Tr. Swiss Ophth. Soc.*, May 4, 1934) on closure of retinal tears by means of cathode electrolysis. Vogt's catholysis is by far the most considerate of the eye tissues, as comparatively small amounts of electrical energy are introduced into the eye. Accordingly, this method is much better suited to a delicate tissue like the retina than are the much more radical and energetic procedures. Hence there is an agreeable absence of unpleasant complications such as secondary tears and late hemorrhages. Furthermore, the loss of vitreous is slight, even if several dozen punc-

tures are made in the course of a single session. The strength of the current should not exceed from 0.5 to 1 milliampere, and the needle should only just penetrate. The method is particularly applicable to the treatment of extensive or multiple tears. The needle-pricks must hit the margins of the tear and its floor as well. The process can be checked up exactly with the ophthalmoscope by observing the generation of hydrogen bubbles when the cathode is applied. In comparison with operations employing the trephine, the short time required for catholysis is worth noting. Clinical results with this procedure up to the present have been uniformly very good. Von Szily and Machemer have also reported on the results of their trial of unipolar electrolysis as well as of bipolar surface electrolysis.

Dr. von Szily, Münster: The technic and the effect of both unipolar and bipolar electrolysis for the localization and closure of retinal tears have been described in collaboration with Machemer in two publications in the *Klinische Monatsblätter für Augenheilkunde* and previously to that in two papers read before the Association of Rhein-Westphalia Ophthalmologists. The first of these communications dates back more than a year. It calls attention to the good results of this method in human detachment and particularly to the delicate nature of the scars produced by bipolar surface electrolysis. It is most gratifying that Dr. Vogt accepted this procedure at my suggestion and urging, and obtained satisfactory results with it just as I did.

Dr. Lindner, Vienna: The frequency of vomiting in Meesmann's cases is probably to be explained by the fact that instead of cutting through the muscles he drags them aside rather forcibly. The percentage of cures in my clinic mentioned by Gilbert refers to the entire number of cases in which operation was performed. My associates and I operated in practically every case that presented itself. The proportionate number of cures would have been decidedly higher if we had excluded the unfavorable cases. My article published in 1931 gave an explanation of the less favorable operative prognosis in cases of detachment with extensive or deep bulla. Gonin's methods should no longer be used, because if one does not succeed at the first operative attempt, the eye thus treated can only very exceptionally be subjected to any further surgical interventions with a prospect of success. With the newer methods, any subsequent operations which become necessary attain a much larger number of reattachments. The most important consideration in all operations is, in my opinion, the limiting of fistulization points to the supraretinal space. One should never open into the vitreous, which is itself detached in most cases of spontaneous retinal detachment.

Dr. Larsson, Stockholm: In regard to vomiting as a complication of operations for detachment, I should like to make a few remarks on the following points: I do not use retrobulbar injections but, in spite of this, I often have to deal with vomiting on the part of the patient. They say that in England the ophthalmic surgeons are not troubled with this particular complication. I cannot explain this marked difference in the reaction of patients who, if one may judge from the reports, are prepared for operation and anesthetized there just as they are in Germany. I used to detach the muscles by section but do not do so any longer, and yet vomiting occurs just as often as it did before. Against this complication I treat patients with atropine and scopolamine by

mouth and have excellent results. In reference to the paper read by Engelking, in the case of detachment with a deep and extensive bulla I direct that the patient be put to bed for several days. At operation, I start with a surface coagulation and only after that proceed to trephining. Possibly this is not the most suitable procedure. I have the impression, as Engelking has, that the combination of surface diathermy with multiple diathermic punctures might be more profitable, but, as mentioned previously, I have so far worked only with surface diathermy, as I wished to familiarize myself with all the details of this procedure and learn all there was to be known about it before taking up any different form of operation or modifying the technic in any way. Answering Dr. Bartels, I wish to say that in my operations for detachment I always trephine whether the detachment is deep or shallow.

Dr. Meesmann, Berlin: Mechanical insult to the muscle by pull cannot be the cause of the vomiting, as it does not take place when one uses n-methyl-cyclo-hexanyl-methyl barbituric acid for narcosis. The suture thread passed through the muscles and looped as a bridle greatly facilitates ophthalmoscopy, as an assistant can easily put the eye in any desired position by pulling on these retractors. The operation begins with a surface coagulation. This is followed by the insertion of fine needles to produce areas of coagulation at the margins of the tear, and then, toward the last, coarse needles to allow the subretinal fluid to drain off. In operations for relapse, use of the fine needle is to be preferred to surface coagulation.

The Anterior Segment of the Eyeball in Renal Disease.

Dr. Mylius, Hamburg.

Very slight attention has been paid in the past to the point indicated by the title, although in the last decade it is just the reciprocal relationship between eye disease and kidney disease which has been made the subject of frequent and active discussion. Clinically it is true that even the severest nephritis shows comparatively few signs in the anterior segment of the eye if one excepts the well known conditions of edema affecting the ocular adnexa. However, a more careful examination and especially a routine use of the slit lamp will show in almost all cases of severe renal disease a more or less diffuse, although slight, clouding of the aqueous or even corpuscular elements in more concentrated suspension. Systematic microscopic study of histologic sections of the anterior segment of the eyeball was carried out in 32 eyes of subjects who had renal disease from my own large material and on 54 eyes kindly placed at my disposal by Professor Hanssen.

Such a study could not fail to throw light on the anatomicopathologic changes at the anterior pole of the eye in renal disease and might possibly give some new data which would tend to clear up the pathogenesis of the most important morbid ocular changes, especially of albuminuric retinitis. Basing the study of this large material on serial sections, my collaborators and I were able to show that in nephritic persons even of a tender age there is often a fatty infiltration into the cornea, the sclera, the structureless tissues and the subepithelial villi, besides proliferation of connective tissue with sclerosing changes throughout the entire uveal tract which was primarily and principally prominent in the neighborhood of blood vessels. Especial attention was paid to the vascular

system of the anterior segment. In marked contrast to Ginsberg's view that arteriosclerotic changes in the ciliary body are very rare and, in renal disease, are limited to the posterior segment, it was found that such vascular changes do take place in the anterior part of the choroid and in the ciliary body. It was only in the iris that they were not found, and in this region, too, no fatty infiltration of the walls of vessels could be demonstrated in a single one of the many serial sections of the iris which were specially stained with osmic acid. More or less marked lesions were found in the vessels of the ciliary body and in those of the anterior portion of the choroid in 21 cases. No pathologic changes were found in the vessels of the extreme periphery of the retina. A finer and more detailed analysis of the histologic changes in the vessels of the anterior segment showed, besides slight, incipient proliferation of the intima, advance in that pathologic process to the highest degree and at times complete occlusion of the vessel. There were also simply sclerosed vessels and others with hyaline degeneration, some with marked hypertrophy of the muscular coat, extreme lipoid infiltration, as well as disorganization of the cells of the wall of the vessel into droplets, and far advanced cell degeneration. In most cases the vascular changes in the anterior segment of the eye are not to be considered as a part of a generalized systemic arteriosclerosis, but stand in a causal relation with the general vascular disease which is concurrent with the disease of the kidney, as is known in regard to the pathologic changes in the uveal tract. These observations give ground for serious objections to Koyanagi's theory of the pathogenesis of renal retinitis, which is based on the assumption, which this study proves to be incorrect, that the morbid changes in the choroid and choroidocapillaries are limited to the posterior pole.

DISCUSSION

DR. GILBERT, Hamburg: My original analogy was expressly not that of nephritis to iritis but of nephrosclerosis to iridosclerosis. This parallel was intended to bring out the point that histologic changes may take place in the kidney as well as in the iris which in both instances should be included in the category of arteriosclerosis. I myself had a few histologic data as to the ciliary body, and even as to the iris in a case of a not so old patient with nephritis. I did not go into further detail as to these observations at the time, as the object of my purely clinical communication was to show that changes in the vessels of the iris might well come into consideration in the pathogenesis of postoperative circulatory disturbances or inflammatory processes. To judge by the pictures of sections which have just been shown, one will have to deal less with changes in the vessels of the iris than with those in the vessels of the ciliary body. But that the former also occasionally come into question as a cause of postoperative inflammations must readily be conceded. As a matter of fact, what one often calls "iritis" for short, is an iridocyclitis. The fact remains that in the twelve years which have passed since I read my paper at the meeting in Vienna I have seen 2 cases of postoperative inflammatory complications in patients in the eighth and ninth decade of life in which no other cause could be found, as the operation had been performed with perfect technic and was not followed by either endogenous or exogenous infection; besides which, in one case, the extraction had been made in the capsule.

BACILLEMIA IN OCULAR DISEASE. DR. VON HIPPEL, Göttingen.

The study of this subject with the aid of blood cultures carried out by Löwenstein goes back two and a half years and involves 116 cases, 27 of which were reported to have yielded positive results and 89, negative results. The clinical diagnosis was not communicated to Löwenstein until after his report on the bacterial content of the blood had been received. The finding of bacilli in the blood has the significance of an important addition to diagnostic methods. While it is not decisive in the diagnosis of ocular tuberculosis, it certainly increases the probability that the disease is tuberculosis, and that to a marked degree. Tissue cultures are even more convincing, but can be made only in exceptional cases. The cases investigated could be arranged in six groups: (1) iritis and iridocyclitis, 46 cases, in 12 of which the blood yielded bacilli; (2) sympathetic ophthalmia, 1 case, in which the blood culture was negative but in which there was an unusually marked intradermal reaction and a favorable course under treatment with a tuberculin preparation; (3) retinal perivasculitis, 12 cases, all of which yielded negative blood cultures, but 10 of which presented strongly positive reactions to tuberculin; (4) retrobulbar neuritis and papillitis, 19 cases, in 6 of which the blood cultures were positive; (5) a group including scleritis, deep and phlyctenular keratitis and choroiditis, 27 cases, in 6 of which the blood cultures were positive; (6) penetrating injury, 11 cases, in 3 of which the blood cultures were positive. There is correspondence to a marked extent between the finding of bacilli in the blood and the positive results of diagnostic methods previously used. But the cases in which the blood cultures were negative correspond in the main, as to later clinical course and reaction to tuberculin, to the cases in which such cultures were positive, so that failure to find bacilli in the blood does not speak in any way against the diagnosis of tuberculosis. The 3 positive cases of penetrating injury were not, as may have been surmised, complicated by sympathetic ophthalmia. So far, no support has been found for the assumption that patients whose blood yields the bacilli are especially susceptible to the hazard of sympathetic ophthalmia, as could theoretically be expected. Besides Löwenstein's 3 cases there was one quite similar in which Kolle reported positive results and in which the eye was enucleated but in which histologic examination did not give the slightest support to a diagnosis of sympathetic uveitis.

DISCUSSION

DR. ARRUGA, Barcelona, Spain: Twenty-four blood serums were examined simultaneously at Vienna and at Barcelona. They were from patients whose condition clinically was more than suggestive of tuberculosis, and so the serums could have been expected almost surely to show positive findings. The serums from patients with such conditions as sclerosing keratitis and relapsing phlyctenulae were invariably negative, as were the serums from 2 patients with sympathetic ophthalmia.

EXPERIMENTAL INOCULATION OF FOWL AND APES WITH MATERIAL FROM HUMAN SYMPATHETIC OPHTHALMIA, WITH REMARKS ON THE NATURE OF THE EXCITING AGENT. DR. A. VON SZILY, Münster.

I started with the assumption that human sympathetic ophthalmia depends on an infectious process of a special nature and instituted

experimental studies which followed closely the results of my model experiments with herpes virus (1924) to produce experimentally an endogenous transmission of infection from eyeball to eyeball. The material used was uveal tissue from patients with true sympathetic ophthalmia. The experiments gave a definitely positive result inasmuch as a hitherto undescribed and specific finding was made not only in the animals originally inoculated but in the second series inoculated from those, that is, in the second generation of inoculations communicated from animal to animal and even from one species to another. The most striking feature histologically was the appearance of masses of round cells which at some points had the structure of true lymph follicles. Apparently one dealt with products of tissue reaction which so far are proper to this infection alone. The way in which the virus (*Noxe*) spread to the surrounding tissue, to the contents of the orbit, to the optic nerve, the chiasm and the optic tract, and also to the nerve, orbit and eyeball of the opposite side, reminds one in many ways of the data on the transmission of herpetic infection from eye to eye. There is, however, a marked difference besides the specific nature of the histologic products of the reaction, in that the virus of sympathetic ophthalmia, in contradistinction to the virus of herpes, is nonpathogenic for the cornea, does not spread diffusely in the central nervous system and does not cause encephalitis or, as a matter of fact, the slightest constitutional symptoms of any kind. This histologic picture does not correspond completely, it is true, to that of human sympathetic ophthalmia, although the tissue changes themselves are very typical. To begin with, the process runs a much milder course in the second eye, and this is most probably the reason why it has been assumed that it is extremely difficult to produce sympathetic ophthalmia in animals by inoculation and that the condition probably never occurs spontaneously. In the present experimental series, neither traumatization of the ciliary body nor intravenous superinfection with tubercle bacilli brought about any change in the reaction of the animals to inoculation.

Quantitative Determination of Urea in Normal and in Pathologic Lenses. Dr. Grümer-Schmoll, Giessen.

In a recent work on ophthalmology, that of Schieck and Brückner (*Kurzes Handbuch der Ophthalmologie*, Berlin, Julius Springer, 1932, vol. 2), it is stated that urea has, so far, not been found in the lens. However, the very latest studies, two in number, have shown, at least qualitatively, the presence of that substance, so that it was thought worth while to work out a simple method for the quantitative determination of urea. The best method was found to be that of Fosse with xanthydrol, in which dixanthyl-urea crystals are formed, and the amount of urea is computed from their weight. These investigations dealt with lenses of human beings, swine, rabbits, cattle and cats. It was found that in animals clear lenses contain large quantities of urea, especially in the early years, and that these quantities grow less and less with advancing age. Rabbit lenses showed a urea content of from 40 to 50 mg. (sic) per hundred grams of tissue in youth and from 6 to 8 mg. in old age. A similar diminution in the urea content is noted in cataractous lenses in man. The determinations could be carried out only on cataractous lenses on account of the difficulty of getting sufficient

material in the shape of clear lenses, obtained either immediately post mortem or after enucleation. In the opaque lenses there was still a urea content of about 20 mg. per hundred grams of tissue. But here the comparison with clear human lenses was lacking. The object of the paper has been to popularize a simple quantitative test for urea in individual lenses and to urge further studies of lenses after enucleation of eyes or after severe ocular injuries, in order to throw further light on the normal and on the pathologic metabolism of the lens.

STUDIES IN LENS METABOLISM. DR. MÜLLER, Basel, Switzerland.

Research into the significance of vitamin C gave rise to the inference that quite possibly the lens itself produces vitamin C. The following experiments give several points of support for this assumption. If an ox lens is kept in its own aqueous at a temperature of 37 C. in the incubator for five hours, a slight but distinctly noticeable increase takes place in the vitamin C content of the lens, averaging 5 mg. per hundred grams of tissue. This experimental result is probably to be ascribed to increased production of vitamin C in the lens, presumably from dextrose. For it can be demonstrated that the lenses of older oxen, normal or cataractous, show in comparison with the lenses of calves, not only a lowering of the vitamin C content, but of the sugar content as well. Calves' lenses generally contain, on an average, 115 mg. of sugar and 39 mg. of vitamin C. per hundred grams of lens; clear lenses of grown beeves, 73 mg. of sugar and 29 mg. of vitamin C; opaque ox lenses, 36.4 mg. of sugar and 6 mg. of vitamin C. Evidently, sugar content and vitamin C content show directly similar and parallel changes in aging and in the development of cataract. The conditions in the aqueous are somewhat different. The aqueous of calves' eyes contains, on an average, 58 mg. of sugar and 15 mg. of vitamin C, whereas that of older animals contains, on an average, respectively, 48 and 19 mg. The experiments show further that the aforementioned increase in the production of vitamin C is accompanied by a marked decomposition of sugar in the lens. In the incubator after five hours under the conditions mentioned, there is an average loss of 20 mg. of sugar per hundred grams of lens. This substance and vitamin C seem to be closely linked in the lens.

CONTUSION CATARACT. DR. BÜCKLERS, Tübingen.

I have seen the rosette form of lens opacity, which comes on some time after injury and was first noted and described by Vogt under the name of *traumatische Spaetrosette,* in 45 cases of cataract in the eye clinics at Berlin and Tübingen. Only 10 per cent of the patients were females. The number, form and distribution of the leaf-shaped opacities varied greatly. It was possible to examine 5 of these patients shortly after their injury. Their lenses showed faint star-shaped subcapsular opacities. The so-called late rosette, which, as its name implies, does not come on until a long time after injury, appears in two different types. In one, which is the commoner of the two, the leaves are narrow and sharp. The other presents broad leaves, which run out toward the periphery of the lens. Examples of particularly characteristic types and developmental stages were demonstrated. With advancing age, the contusion rosette goes down deeper and deeper into the substance of

the lens. Its position in relation to a given zone of discontinuity offers, accordingly, definite criteria as to its age and allows one to draw far-reaching conclusions as to the date of the accident. This point is of special practical importance in case a subsequent less serious injury has taken place, to which the patient now attributes his loss of vision. The differentiation between zonular cataracts and various other forms of lens opacity is discussed. In general, a statistical study showed that, of 45 patients, only 4 knew nothing about having suffered an injury. Accordingly the conclusion is reached, contradicting Handmann, that the late rosette never develops except on the basis of a previous traumatism. Three other patients presented a quite different form of cataract, one which, so far, seems to have been scarcely recognized, coming on, as it does, after a special form of indirect injury, namely, that from an explosion. In such cases one finds numerous sharply defined white dots arranged predominantly along the sutural lines of the lens star figure. This form of traumatic cataract can be compared only to those pictured by Vogt as glaucomatous white dots of the lens (Atlas of the Slitlamp-Microscopy of the Living Eye, translated by Robert Von der Heydt, Berlin, Julius Springer, 1921, pt. 2). Hence, one deals with a rare form of contusion cataract which may well be due, as are the glaucomatous punctate opacities of the lens, to a sudden increase of intra-ocular tension.

EXTRACTION OF DISLOCATED LENSES WITH THE HIGH FREQUENCY NEEDLE. DR. JESS, Giessen.

Three cases are reported in which the operation was performed successfully. The first case was one of traumatic dislocation of the lens into the vitreous with secondary glaucoma; the second, one of congenital dislocation with cataract, in which it was particularly worth noting that even this juvenile lens came out complete in the capsule; the third was one of dislocation of the lens into the anterior chamber with secondary glaucoma. In a fourth case the extraction of the dislocated lens was only partially successful, and a second operation had to be performed. In view of the scarcity of clinical material bearing on this operative procedure, it is hoped that further experience will be collected and communicated. In conclusion, it is pointed out that this coagulation extraction with the needle can be put to good use if, in the course of an Elschnig intracapsular extraction, one is confronted with a rupture of the capsule and the body of the lens remains between the lips of the section. The electric needle, if introduced at once, will permit the completion of the extraction much better and with less danger than the loop and/or forceps.

DISCUSSION

DR. ENGELKING, Cologne: I was able to use Jess' method, but only in a single case, and with complete success. As the lens had sunk deep down into the vitreous, a long and very fine needle was used, well insulated except at the tip which was bent to a right angle. This enabled one to "grasp" the lens from behind before the current was turned on and so to avoid the danger of pushing the lens still farther away with the point of the needle in attempting to establish contact. Extraction was completed without loss of vitreous, so that Jess' method can be warmly recommended.

Dr. Jess, Giessen: I might mention another modification of my procedure suggested by Tobgy of Egypt. The tip is fastened into a small plate with three small hooks. It is possible that at times the lens can be grasped more easily and firmly with this instrument than with the simple needle tip. Unfortunately (sic) such dislocations of the lens are too rare for one to get an extensive experience in handling them. It would be most welcome to have those practitioners who have so far not practiced the procedure report any cases which come under their observation. In reply to Müller, it is noted that he was able, with vitamin C, to bring about a marked bleaching of the iris pigment in rabbits' eyes. After the injection of a small amount of ascorbic acid into the anterior chamber the previously brown iris became pale blue by depigmentation. Histologic research into this surprising reaction has not been carried out so far, as the observation is of recent date. The bleaching action of vitamin C is certainly interesting and may well have some bearing on the question of the etiology and pathogenesis of heterochromia of the iris.

(*To be concluded*)

Book Reviews

Coincident Ophthalmoscopy and Histology of the Optic Nerve. By Dr. Gustav Guist, Vienna. Price, 250 Austrian schillings. Pp. 169, with 164 illustrations. Privately published.

This is the work on which, as many ophthalmologists have known, Dr. Guist has been engaged for the past five years. The idea expressed in the title of demonstrating the pathology of lesions of the fundus by the presentation of pictures and histologic sections together must have existed as a vague dream in the minds of many ophthalmologists. The late Dr. Finnoff, in his lectures on retinal pathology, made some use of this idea, as did Dr. Klien and Dr. Moncrieff in a recent exhibit, and very effectively. It remained for Dr. Guist to demonstrate completely its possibilities in the present book.

The book is a collection of one hundred and sixty-four illustrations, most of which are in color, with an accompanying text. A few of the pictures of the fundus are photographs made in color by the multiple filter method. Most are colored paintings made by Professor Dietz. In regard to execution and reproduction these are impeccable. With the pictures of variations in the normal nerve head and the principal pathologic conditions are photomicrographs illustrating the underlying pathology. These are reproduced in color by a new photographic process so as to resemble the actual stained sections more nearly than any reproductions which the reviewer has seen.

The material has been chosen with such care that it requires no effort of the imagination to believe that the condition of the fundus is actually represented. In many cases this is placed beyond doubt by the presentation in sections of the same eye of which the picture of the fundus was made. This is true, for example, in the cases of myopic conus, inferior conus, temporal pigment ring, Tay-Sachs disease, embolism of the central artery, thrombosis of the central vein and drusen of the optic papilla. Various stains were used, which are reproduced well.

No more perfect means could be employed to make one understand the reason for various pictures seen ophthalmoscopically. It is shown, for instance, that the opacity of the retina in embolism of the central artery is due not to edema but to necrobiosis of the retinal elements, especially the third neuron with its ganglion cells. The drusen of the optic nerve and of the lamina vitrea are shown to be different, the former being hyaline, while the latter are composed of a different substance formed by the pigmentary epithelium. A picture of the fundus in pseudoneuritis is shown, with the histologic demonstration that it is due not to an accumulation of glia on the disk, but to the formation of the optic canal, which is straight and narrow, so that the nerve fibers are forced to bulge forward on the nerve head for lack of space in the canal. These are only a few of the considerations which will stimulate a more careful interpretation of ophthalmoscopic findings

by the experienced ophthalmologist, while for the beginner the study of this book will put his ophthalmoscopy, from the start, on a rational basis different from the usual method of attaching descriptive names to conditions which are not understood.

The text is in English by the author, no German edition having so far been printed. It is in general free from Germanisms, and there are remarkably few misprints. No references are given. While, for the most part, one is content to accept the author's explanations of phenomena of the fundus from the evidence of his material, in some instances one would be interested in knowing a little more of the literature. A number of these explanations seem to be original with the author. There are a few subjects still open to controversy, which are dismissed in a somewhat dictatorial manner. Thus, the fact that papilledema may be due to sinus disease is stated without argument, and the theory of papilledema accepted by the author is given without qualification. Dr. Guist believes that this condition in arterial hypertension is always due to increased intracranial tension. Apparently he ignores the important work of Verhoeff showing that most tumors of the optic nerve are true gliomas, classifying them as endotheliomas, for the most part. He adheres to the unfortunate name glioma retinae for what should be called retinoblastoma, without discussion of recent observations with neurohistologic methods, which explain the nature of the characteristic rosettes. It is in the chapter on tumors that one most misses some discussion of the literature.

As if the labor previously described were not enough, the author has published the volume himself, which necessitated provision of machinery for reproducing the new color process and actual production of the illustrations.

On the whole, the book is a superb contribution to knowledge of the pathology of the fundus. When the number of illustrations and their excellent quality is considered, its price seems far from excessive. It is to be hoped that a further volume will be devoted to conditions of the choroid and retina.

SANFORD R. GIFFORD.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217 Faubourg St. Honoré, Paris.
Secretary-General pro tem: Dr. A. Churchill, 66 Boulevard St. Michel, Paris.

International Ophthalmologic Congress

Secretary: Dr. Francisco Poyales, Olozaga 3, Madrid.
Place: Cairo. Time: 1937.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. A. J. Ballantyne, 11, Sandyford Pl., Glasgow, C 3.
Secretary: Dr. J. Ringland Anderson, 108 Collins St., Melbourne.
Place: Melbourne. Time: Sept. 11-13, 1935.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. R. Foster Moore, 53, Harley St., London, W. I.
Secretary: Miss Ida Mann, 7, Wimpole St., London, W. I.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford. Time: July 4-6, 1935.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.
Secretary: Dr. J. D. Magor Cardell, 27, Weymouth St., London, W. 1.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

Society of Swedish Ophthalmologists

President: Prof. F. Berg, Uppsala, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.

Chinese Ophthalmological Society in Peiping

President: Dr. P. H. Liu.
Secretary: Dr. H. H. Feng.
Place: Peiping Union Medical College, Peiping.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan.
Place: Cheeloo University School of Medicine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Arthur J. Bedell, 344 State St., Albany, N. Y.
Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Place: Atlantic City, N. J. Time: June 10-14, 1935.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Wells P. Eagleton, 15 Lombardy St., Newark, N. J.
President-Elect: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Cincinnati. Time: Sept. 16-20, 1935.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Walter B. Lancaster, Boston.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Hot Springs, Va. Time: June 5-7, 1935.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Benjamin Sachs, 483 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Frank B. Kistner, Mayer Bldg., Portland, Ore.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Portland, Ore. Time: May 20-23, 1935.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Francis A. Brugman, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Noble Dick, 1115 Terry Ave., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Harry Shearer, 405 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. W. S. Beyer, 1101 Talcott Bldg., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Charles H. Baker, 805 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. J. Hompes, 1307 N St., Lincoln, Nebr.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.
Place: St. Louis. Time: November 1935.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. F. B. Fralich, 203 S. Main St., Ann Arbor, Mich.
Secretary-Treasurer: Dr. A. S. Barr, 201 S. Main St., Ann Arbor, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Claude W. McKee, 229 S. Main St., Greensburg.
Secretary-Treasurer: Dr. C. W. Beals, 41 N. Brady St., DuBois.
Place: Indiana. Time: October 1935.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 227, 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Arthur M. Yudkin, 257 Church St., New Haven.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. Herschel C. Crawford, 26 Lindon Ave., N. E., Atlanta.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.
Place: Atlanta. Time: May 7-10, 1935.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Ora G. Brubaker, North Manchester.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.
Place: Indianapolis. Time: April 10, 1935.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. W. Dean, 532, 1st Ave., Council Bluffs.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Council Bluffs. Time: 1935.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. John W. Orr, 503 S. Saginaw St., Flint.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. James A. Reynolds, 78 S. 9th St., Minneapolis.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Lawrence G. Dunlap, 101 Main St., Anaconda.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. James A. Fisher, 501 Grand Ave., Asbury Park.
Secretary: Dr. William K. Campbell, 96 Third Ave., Long Branch.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Webb W. Weeks, 20 E. 53rd St., New York.
Secretary: Dr. John F. Fairbain, 925 Delaware Ave., Buffalo.
Place: Albany. Time: May 13-15, 1935.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. W. R. Winn, 702 First Ave., S., Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.
Place: Minot. Time: June, 1935.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary and Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Josiah E. Smith, 60 Meeting St., Charleston.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Eugene Orr, 706 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Broad St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. William Stookey, 54 E. South Temple St., Salt Lake City.
Secretary-Treasurer: Dr. T. F. Welsh, 711 Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Beverley R. Kennon, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. George G. Hankins, Medical Arts Bldg., Newport News.
Place: Fredericksburg. Time: May 4, 1935.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. W. F. Beckner, 1050 Fifth Ave., Huntington.
Secretary: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Place: Wheeling. Time: May 1935.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Alfred Stahl, 55 Lincoln Park, Newark, N. J.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Herschel Crawford, 478 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. Stacy C. Howell, 384 Peachtree St., N. E., Atlanta, Ga.
Place: Academy of Medicine, 13 Queen's Park. Time: 8 p. m., last Thursday of each month.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Lloyd B. Whitham, Mt. Vernon Pl. and Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. John H. Bailey, 855 St. Marks Ave., Brooklyn.
Secretary-Treasurer: Dr. George Freiman, 703 Grand St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. H. H. Glosser, 448 Franklin St., Buffalo.
Secretary-Treasurer: Dr. I. J. Koenig, 40 North St., Buffalo.
Time: Second Thursday of each month.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. E. V. L. Brown, 122 S. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Thomas D. Allen, 122 S. Michigan Ave., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.
Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

CLEVELAND OPHTHALMOLOGICAL CLUB

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.
Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. John Milton Griscom, 2213 Walnut St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. W. C. Davis, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. H. M. Sage, 9 Buttles Ave., Columbus, Ohio.
Place: Athletic Club. Time: First Monday of each month.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. W. Mood Knowles, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. L. H. Quinn, 4015 Live Oak St., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. C. Walker, 410 6th Ave., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Secretary-Treasurer: Dr. W. C. Mott, 214 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. T. L. Goodman, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. John H. McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 307 Metz Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. R. M. Hargrove, 1202 Esperson Bldg., Houston, Texas.
Secretary: Dr. J. C. Dickson, 1617 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. C. H. McCaskey, 404 Continental Bank Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Raymond E. Teall, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. A. W. McAlester, 2003 Bryant Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Russell T. Uhls, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Kenneth C. Brandenburg, 707 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. F. H. Brandt, 1136 W. Sixth St., Los Angeles.
Secretary-Treasurer: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. E. A. Leggett, 614 Breslin Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. E. Craddock, 604 Breslin Bldg., Louisville, Ky.
Place: Jewish Hospital. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. R. S. Pendexter, 900, 17th St., N. W., Washington.
Secretary: Dr. Lyman B. Tibbets, 1801 Eye St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, 130 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. J. J. Brook, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. Herbert G. Schmidt, 238 W. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6 p. m., second Tuesday of each month.

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Secretary: Dr. A. L. Guertin, 410 Sherbrooke St., West, Montreal, Canada.
Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Wesley Wilkerson, Bennie Dillon Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. Hale Cullom, Bennie Dillon Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Webb William Weeks, 20 E. 53d St., New York.
Secretary: Dr. Guernsey Frey, 121 E. 60th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. John H. Judd, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Robert H. Rasgorshek, 425 Aquila Court, Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Secretary: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward B. Heckel, 500 Penn Ave., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

PITTSBURGH SLIT-LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Samuel M. Cottrell, 116 E. Franklin St., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Laurence Macey, 221 Alexander St., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Meyer Wiener, 308 N. 6th St., St. Louis.
Secretary: Dr. W. M. James, Metropolitan Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. Frank Steed, Nix Professional Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Frank H. Rodin, 490 Post St., San Francisco.
Secretary: Dr. Louis F. Morrison, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Secretary-Treasurer: Dr. Leon F. Gray, 1214 Slattery Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 8:15 p. m., fourth Tuesday of every month except June, July and December.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Marc Anthony, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. Raymond A. Lower, 1455 Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. D. F. Gillette, 109 S. Warren St., Syracuse, N. Y.
Secretary-Treasurer: Dr. W. J. Werfelman Jr., 725 State Tower Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Clarence E. Hill, 160 Bloor St., West, Toronto.
Secretary: Dr. A. Lloyd Morgan, 86 Bloor St., West, Toronto.
Time: First Monday, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. William Thornwall Davis, 927 Farragut Sq., Washington, D. C.
Secretary-Treasurer: Dr. James N. Greear Jr., 1740 M St., N. W., Washington, D. C.
Place: Episcopal Eye and Ear Hospital. Time: 8 p. m., first Thursday in November, January, March and May.